Medical Parasitology in Tables

2nd Edition

Dr. Azza Al-Adawi (supervisor)

Osama Esam

Omar Aldurini

Ibrahim Sehsah

This work is meant to help our friends in their medical course as undergraduates. We hope that this work will benefit all of them. So, please do not forget us in your do3aa.

We depend on more than one source to get the information, but in the limits of our course as 3rd year medical students and the main source is the department book. There is no new information in this work but the arrangement of the information and some notes. We hope that it will help anyone who needs help as we did.

Thanks to Dr. Azza for her time to review and put some comments on this work. Our best wishes for her are indescribable. Do not forget her in your do3aa too.

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PLEASE NOTE (N.B.):

a. ***This paper of work is not a 100% perfect information source, although we hope it is.***
b. ***Do not rely on this work (only) in study of medical Parasitology. This is just a bit of work to help you in your medical course.***
c. ***If you find any wrong information in here or you have an idea about it, please send any of us a message to the email or account or call us directly.***
d. ***This paper work is free to anyone. Please do not try to sell it anyway.***
e. ***The normal size of this paper is A3.***
f. ***Do not forget us in your do3aa.***

How to study medical Parasitology?

1. General characters & life cycle: read & understand.
2. General Distribution: Enough to know whether the parasite is present in Egypt or not (very important in cases).
3. Pathogenesis, Clinical picture and diagnosis: very important study them very well.
4. Treatment: enough to study the first drug written in your book (it is called the drug of choice) NO NEED to study any doses for drugs.

Class: Trematoda

General characters:

1- Flattened dorso-ventrally, bilaterally symmetrical and unsegmented.
2- Provided with suckers: an anterior oral and ventral suckers, sometimes a third genital sucker.
3- Having a protective cuticle either smooth or provided with spines or tubercles.
4- Muscle fibers: longitudinal, circular and oblique help in the movement of the parasite.
5- Nervous and excretory systems are present but there is no respiratory or circulatory system.
6- The digestive system starts by mouth surrounded by oral sucker anteriorly. This leads to a short pharynx that bifurcates in front of the ventral sucker into two intestinal caeca that may be simple or branches and end blindly.
7- The genital system hermaphrodite (except Schistosoma) having both male and female reproductive organs in one worm. Usually there is cross-fertilization between 2 separate adults but self-fertilization may occur.
8- General life cycle: adults → eggs → water → miracidium → snail → Sporocyst → redia → cercaria → infect man → adults.

Parasitic pharyngitis:

In Lebanon & America, people have a habit of ingestion of fresh raw sheep & goat livers. If these livers are infected with Fasciola, living worms will attach to the pharyngeal mucosa causing: oedematous congestion of the pharynx, soft palate, larynx, nasal fossae and Eustachian tubes (suffocation known by the natives as Halzoun). Another cause for this condition is tongue worms (Linguatula serrata).

Mode if infection:

Infection occurs by ingestion of nymph stage in improperly cooked sheep viscera.

Treatment:

1- Gargling with strong alcoholic drinks.
2- Administration of emetics.
3- Tracheostomy in laryngeal obstruction.

Control:

Proper cooking of animal tissues.

General characters of schistosoma:

1- The adults have separate sexes.
2- The two sexes are dissimilar in appearance.
3- The adult worms parasitize blood vessels.
4- They lack a muscular pharynx and the two intestinal caeca reunite into a single caecum.
5- They produce non-operculared eggs.
6- The cercaria, with forked tail, invades the final host percutaneously.
7- No redia stage.

Classification of Helminthes

Platyhelminthes (Flat worms)	Flukes / Trematoda (Disease)	Liver fluke	Fasciola hepatica/gigantica (Fascioliasis)
		Lung fluke	Paragonimus westermani (Paragonimiasis)
		Intestinal fluke	Heterophys (Heterophysiasis)
		Blood fluke	Schistosoma Haematobium, Mansoni & Japonicum (Schistosomiasis) (Swimmer's itch)
	Tapeworms / Cestoda (Disease)	Pseudophyllidea	➢ Diphyllobothrium Latum (Diphyllobothriasis) ➢ Diphyllobothrium Mansoni & Proliferum (Sparganosis)
		Cyclophyllidea	➢ Taenia saginata (beef) (Taeniasis saginata) ➢ Taenia solium (pork) (Taeniasis solium/Cysticercosis) ➢ Echinococcus granulosus (Hydatid Disease or Hydatidosis) ➢ Echinococcus multilocularis (Alveolar Hydatid Disease) ➢ Multiceps multiceps (Coenurosis) ➢ Hymenolepis nana (Hymenolepiasis) ➢ Hymenolepis diminuta (Hymenolepiasis diminuta) ➢ Dipylidium caninum (Dipylidiasis)
Nemathelminths (Round worms)	Intestinal nematoda	In small intestine	➢ Ascaris lumbricoides (Ascariasis) ➢ Hookworms (Ancylostomiasis): o Ancylostoma duodenale o Necator americanus ➢ Strongyloides stercoralis (Strongyloidiasis) ➢ Trichostrongylus colubriformis (Trichostrongyliasis) ➢ Capillaria philippinensis (Intestinal Capillariasis) ➢ Trichinella spiralis (Trichinosis)
		In large intestine	➢ Enterobius vermicularis (Enterobiasis) ➢ Trichuris trichiura (Trichuriasis)
	Tissue nematoda	Adults	➢ Filaria (Filariasis): o Wuchereria bancrofti o Brugia malayi o Loa loa o Onchocerca volvulus o Mansonella perstans o Mansonella ozzardi ➢ Dracunculus medinensis (Dracunculiasis, Dracontiasis)
		Larvae	➢ Trichinella spiralis (Trichinosis) ➢ Larva migrans o Cutaneous: ▪ Ancylostoma caninum ▪ Ancylostoma braziliense o Visceral: ▪ Toxocara canis ▪ Toxocara cati

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Cercarial dermatitis (Bather's itch or Swimmers' itch):

Schistosome of non-human species can penetrate the skin of man but cannot go beyond the germinal layer.

Clinically:

Dermatitis, irritation, itching, oedema and secondary infection.

Diagnosis:

History of contact with water followed by skin rash.

Treatment:

Anti-pruritics, local and general anti-histaminics, antibiotics for 2ry infections.

Control:

- Snail control
- Avoiding dealing with polluted water.
- Thorough drying of skin to prevent cercarial penetration.

Name Parasite (Disease)	Hepatic Flukes Fasciola (Fascioliasis)	Lung Flukes Paragonimus (Paragonimiasis)	Intestinal Flukes Heterophyes (Heterophysiasis)
Geographical Distribution	Fasciola hepatica: Common in sheep raising areas in Europe, Middle East (particularly Egypt), Central & South Africa. Fasciola gigantica: Common in cattle raising areas in South-East Asia & Africa including Egypt.	Asia (South-East), Africa (Nigeria & Cameron) and South America.	Middle and far east, south Europe and in Egypt in brackish water.
Definitive Host & Habitat	Man in bile ducts of liver.	Man in lungs.	Man in between the villi of the small intestine.
Reservoir Host	Herbivorous animals.	Cats, dogs, pigs & monkeys.	Fish eating animals.
Diagnostic Stage	Immature egg stage: Size: 140 × 70 um. Color: yellowish brown. Shape: oval, operculated & thin shelled. Content: immature ovum.	Immature egg stage: Size: 90 × 55 um. Color: brown. Shape: oval, operculated & thick shelled. Content: immature ovum.	Mature egg stage: Size: 30 × 15 um. Color: yellowish brown. Shape: oval, operculated & thick shelled. Content: mature miracidium.
Intermediate Host	Lymnaea cailliaudi (snail) in case of F.gigantica. Lymnaea truncatula (snail) in case of F.hepatica.	1st I.H.: Semisulcospira snail. 2nd I.H.: Crabs, crayfish or shrimps.	1st I.H.: Pirenella conica snail. 2nd I.H.: Tilapia Nilotica (Bolty) & Mugil Cephalus (Boury).
Infective Stage & Mode Of Infection	Encysted Metacercaria through eating contaminated vegetables or drinking contaminated water.	Encysted Metacercaria through eating insufficiently cooked crabs, crayfish or shrimps.	Encysted Metacercaria through eating improperly cooked or freshly salted fish (less than 10 days, sweet Feseekh).
Life Cycle	Adult worms in bile ducts→Eggs →Faeces→Fresh water → Miracidium → Snail host → Sporocyst → Redia → Cercariae (lepto-cercous) → Out to water → Attach to aquatic vegetables →Encysted metacercariae → Ingestion→ Duodenum→ Excysted metacercariae → Migration through the intestinal wall → Peritoneal cavity→ Liver parenchyma → Bile ducts.	Adult worms in lung →Eggs →coughed with sputum or swallowed & excreted in Faeces → Fresh water → Miracidium → 1st I.H. → Sporocyst → Redia →Cercariae (micro-cercous)→Out to water → 2nd I.H. → Encysted metacercariae → Ingestion → small intestine → Excysted metacercariae → Migration through the intestinal wall →Peritoneal cavity → Penetrate the diaphragm & pleura → Lungs.	Adult worms in intestine→Eggs → Faeces → Brackish water → ingested by 1st I.H. → Miracidium → Sporocyst → Redia → Cercariae (lopho-cercous) → Out to water → 2nd I.H.→ Encysted metacercariae → Ingestion → Intestine → Excysted metacercariae → Become deeply embedded between the villi.
Pathogenesis	**1-** If immature flukes migrate through the liver tissue → destruction, necrosis & haemorrhage of the parenchyma. **2-** Hyperplasia of biliary epithelium and fibrous thickening of the ducts as a result of mechanical obstruction, inflammatory responses & the activity of proline excreted by the flukes. **3-** Periductal fibrosis causes pressure atrophy on adjacent liver tissue. **4-** Minute abscesses can form around eggs trapped in the parenchyma. **5-** Spontaneous healing appears to occur frequently and may result from inflammation and calcification. **6-** Flukes that migrate out of the intestine may lose their way and form ectopic lesions.	**1-** Worms provoke granulomatous reactions that lead to fibrotic encapsulation of the worms. **2-** Duo to aberrant migration, larvae may lodge in ectopic sites (brain, abdomen, skin or heart).	**1-** Light infection may pass unnoticed. In severe infections, irritation may produce superficial necrosis, excessive mucous secretion & hyperplasia of the mesenteric lymph nodes. **2-** Patients may suffer from discomfort, colic pain, mucous diarrhea and eosinophilia. **3-** Sometimes eggs may find their way to the circulation where they go as emboli (ectopic lesions).
Clinical Picture	**1-** Diarrhea & digestive disturbance. **2-** Enlarged tender liver, pain in the right costal margin & sub-sternal pain. **3-** Cholangitis, cholecystitis and obstructive jaundice. **4-** Fever, urticaria, anemia and marked peripheral eosinophilia up to 80%.	**1-** Chronic productive cough with brownish purulent sputum containing streaks of blood and parasitic eggs. **2-** Chest pain. **3-** Eosinophilia (20-25 %). **4-** Pleural effusion may occur.	
Diagnosis	**1-** Clinical signs & symptoms (above) & diet history. **2-** Detection of eggs in Faeces or duodenal aspirate is of limited use, because: a. It is only +ve 3 – 4 months after infection. b. Often eggs are undetectable in chronic phase. c. Spurious infection (False Facioliasis): eggs in stool duo to ingestion of liver of infected animals. The eggs disappear after 1 week of liver free diet. **3-** Immunodiagnostic tests: by ELISA, Immuno-fluorescence & counter immunoelectrophoresis which can detect early & chronic infections & are highly sensitive. **4-** Radiological imaging: ultrasonography, endoscopic retrograde & percutaneous cholangiography.	**1-** Clinical signs (above) and diet history in endemic areas. **2-** Detection of eggs in Faeces or sputum. **3-** Adult worms may be expectorated after treatment. **4-** Immunodiagnostic tests: complement fixation & ELISA detect early & chronic infections. **5-** Plain x-ray of chest & tomography show nodular or ring shadows and cavities.	**1-** Clinical signs (above) & diet history. **2-** Finding the characteristic eggs in the stool.
Treatment	**1-** Triclabendazole. **OR** **2-** Bithionol (Dichlorophenol).	**1-** Praziquantel. **OR** **2-** Bithionol (Dichlorophenol).	Praziquantel.
Prevention & Control	**1-** Mass treatment of infected animal reservoir. **2-** Pure water supply. **3-** Snail control. **4-** Human protection by proper washing or cooking of aquatic vegetations. It is advisable to soak vegetables in water containing vinegar for 5 minutes or to put them in water containing drops of potassium permanganate for 10-15 minutes to kill encysted metacercariae stuck to them.	**1-** Treatment of cases. **2-** Good cooking of crabs, crayfish & shrimps. **3-** Health education. **4-** Snail control. **5-** You should know that pigs & small animals are paratenic hosts that can transmit infection if eaten by man. Paratenic host is a host that harbours the parasite in an arrested state.	**1-** Proper cooking and salting of fish. **2-** Periodic examination and treatment of fishermen. They should avoid defecating in water. **3-** Snail control.

Name/Disease	Blood flukes: Schistosoma / Schistosomiasis		
Classification	**Schistosoma hematobium**	**Schistosoma mansoni**	**Schistosoma japonicum**
Disease	Urinary bilharziasis.	Intestinal bilharziasis.	Intestinal bilharziasis.
Geographical Distribution	Nile valley, Africa, Asia, Middle East, South Europe.	Nile delta, Africa, South America, Middle East.	Far East.
Definitive Host & Habitat	Man in Vesical and pelvic venous plexuses.	Man in inferior mesenteric venous plexus in the region of rectum and pelvic colon.	Man in superior and inferior mesenteric venous plexuses.
Reservoir Host	None.	Monkeys and rodents.	Domestic animals.
Diagnostic Stage	Mature egg stage: Size: 140×60 um. Color: Translucent. Shape: oval with terminal spine. Content: miracidium.	Mature egg stage: Size: 150×60 um. Color: Translucent. Shape: oval with lateral spine. Content: miracidium.	Mature egg stage: Size: 85×65 um. Color: Translucent. Shape: oval with minute terminal curved spine. Content: miracidium.
Intermediate Host	Bulinus Trancatus snail in Egypt.	Biomphalaria Alexandrina snail in Egypt.	Onchomelania species snail.
Infective Stage & Mode Of Infection	Furcocercous Cercariae through penetration of the skin of the D.H., aided by: 1- The surface tension of the drying droplet of water. 2- Proteolytic enzymes secreted from penetration glands. 3- Strong lashing movements of the tail pressing the body into the skin.		
Life Cycle	Male carries the female in its gynaecophoric canal towards the peripheral capillaries→ Eggs→ pass to the lumen of intestine or urinary bladder→ Fresh water → Miracidia→ Snail→ Sporocyst (no redia) →Furcocercous Cercariae→ Fresh water→ Penetrate the skin of D.H. → Lose their tail→	Shistosomula→ Venous circulation→ Migration to the lungs → Heart→ Systemic circulation→ Intrahepatic branches of the portal vein→ Maturation→ Migration to the mesenteric veins or to the Vesical veins→ Put their eggs.	
Pathogenesis & Clinical Picture	**1-**Stage if invasion (1-4 days): Local dermatitis, irritation & rash duo to cercarial penetration. **2-**Stage of migration (3-4 weeks): ❖ Lung: verminous pneumonitis, minute hemorrhages, cough & hemoptysis. ❖ Liver: enlarged and tender. ❖ Metabolic products: result in toxic and allergic manifestations as urticaria, eosinophilia, leukocytosis, fever, headache and muscle pain. **3-**Stage of egg deposition and extrusion (acute stage, 1-2 months): Eggs deposited in the venous plexus escape into the perivascular tissue and finally to the outside with urine or stool → tissue damage & hemorrhage. With extrusion, there are: ❖ With schistosomiasis mansoni & japonicum: ○ Dysentery with blood and mucus in stool. ○ Abdominal pain. ○ In S. Japonicum: there is bloody diarrhea and Katayama fever. ❖ With schistosomiasis hematobium: ○ Terminal haematuria. ○ Frequent micturition. ○ Burning pain. **4-**Stage of tissue reaction (chronic stage, months-years): **a.** Tissue proliferation (delayed-type hypersensitivity): eggs trapped in the tissues → stimulate inflammatory reactions → bilharzial granulomas → reversible obstructive lesions. **b.** Tissue fibrosis (immune-suppression-fibroblast proliferation) → irreversible obstructive lesions → bilharzial nodules, papillomata and sandy patches → egg output is reduced. ❖ In schistosomiasis mansoni and japonicum: The intestinal wall becomes fibrosed, thickened and may be complicated with strictures, sinuses, fistulae and prolapse. Eggs that fail to be fixed to the intestinal wall venules fall in the lumen and swept to the liver. This results in periportal fibrosis, portal hypertension, hepatosplenomegaly, acitis and esophageal varices. ❖ In schistosomiasis hematobium: Bladder: fibrosis, 2ry infection, stones and malignancy. Ureter: stricture, hydro-ureter, hydro-nephrosis, 2ry infection and renal failure. Urethra: stricture and fistula. Genital organs: prostate, seminal vesicles, spermatic cord, vulva and vagina may be involved.	**Acute toxemic schistosomiasis or Katayama syndrome:** - Occurs frequently with S. Japonicum & less commonly with S. Mansoni & very rare with S. Hematobium. - High antigenaemia duo to released soluble egg antigens may cross react with rapidly rising antibodies → circulating immune complexes → severe allergic reactions → Katayama syndrome (acute fibril illness) with deposition of these complexes in different sites. - The patient suffers from fever (may last for several weeks), chills, diarrhea, generalized lymphadenopathy and eosinophilia. **Embolic lesions:** ❖ Liver: periportal fibrosis (common in S. mansoni & S. japonicum & may occur in S. hematobium). This lead to portal hypertension, hepato-splenomegaly, acitis and esophageal varices. ❖ Lung: granulomas in the perivascular tissue, pulmonary arteriolitis, obliterated blood flow, pulmonary hypertension and bilharzial corpulmonale (congestive right-sided heart failure). This commonly occurs in S. mansoni & S. japonicum and less in S. hematobium. ❖ Skin, CNS, pericardium and other organs: eggs embolize to ectopic sites via vascular by-pass. **Blood changes:** - Eosinophilia and leukocytosis. - Anemia: ○ Iron deficiency: duo to haematuria. ○ Hemolytic: duo to hypersplenism.	
Diagnosis	**1-**History of infection and endemicity (living or coming from endemic area) **2-**Clinical picture according to the stage of infection. **3-**Laboratory diagnosis: **i.** Direct parasitological methods: ❖ Detection of S. hematobium eggs in urine by sedimentation methods. Examination of the last drops of urine passed after 15 minutes of physical exercise gives more positive results. Eggs should be examined for viability: living eggs are translucent with intact moving miracidium and hatch in fresh water. Dead eggs are opaque with dark granular contents and negative hatching test. ❖ Detection of S. mansoni or S. japonicum eggs in stool by smear technique or by concentration by sedimentation technique. Kato thick fecal smear is helpful for clinical & epidemiological studies. It is a counting technique for detection of worm burden →	and to assess efficacy of the drug. Rectal swab using a gloved finger lubricated with soap. The material obtained is put on a slide and examined. **ii.** Blood examination: anemia, leukocytosis and high eosinophilia. **iii.** Indirect serological methods: resorted to in late or chronic cases where massive fibrosis of the organs affected prevents the ova from excreta. The most common used tests are: - IHAT -ELISA -IFAT **iv.** A recent direct technique is the detection of the adult Schistosome circulating in serum or urine. They indicate active infection by enzyme immuno-assay & have high specificity & sensitivity. **4-**Cystoscopy, colonoscopy and sigmoidoscopy: Done in chronic cases, when eggs are not obvious by routine way, to detect lesions and take biopsies. **5-**Radiology.	
Treatment	**1**- Praziquantel (biltricide).	**2**- Oxamniquine (vansil).	**3**- Metriphonate.
Prevention & Control	**1-**Mass treatment and follow up of infected persons. **2-**Protection: **a.** Health education, pure water supply, treatment of water canals to be safe, proper sanitary measures as construction of latrines in houses, schools and mosques. **b.** Personal prophylaxis for exposed persons e.g. wearing boots & gloves. **c.** Quick drying of exposed skin on getting out of polluted water and application of alcoholic preparations reduce cercarial penetration. **d.** Use of repellants as dimethyl or dibutyl phthalate or diethyl toluamid to prevent cercarial penetration. **3-**Snail control: Physical methods: changing the environment to become unsuitable for snails to live. - Clearing canals from weeds to deprive snails from food.	- Lining banks of canals with concrete to prevent plant growth. - Double canal system: one canal provides water for 6 months and the other is allowed to dry alternatively. - Increasing the velocity of water by increasing the slopes of canals. - Traps of palm leaves at canal inlets to prevent snails. - Diverting the canal sources from passing through villages. Biological methods: - Introduction of a natural enemy which predates on snails as ducks, birds or snails (Marisa species). - Plantation of some plants toxic to snails as Balanites Aegyptiaca. Chemical methods (molluscicides): - Copper sulphate 10 – 20 parts per million. - Sodium pentachlorophenate (santobrite) 5 – 10 parts per million. - Bayluscide 2 parts per million.	

Class: Cestoidea
Subclass: Cestoda

General characters:
1- Flat, ribbon shaped and segmented, hence called tape worms.
2- Covered by cuticle.
3- No body cavity, various systems are embedded in parenchymatous tissue.
4- No digestive system. They feed by diffusion through the cuticle.
5- Any cestode is formed of scolex (head), neck and strobila (chain of segments).
6- Excretory and nervous systems are present.
7- Genital system: hermaphrodite; each mature segment contains male and female genital systems. Common genital pores are either ventral or lateral.

Adult intestinal cestodes are:
D. latum, T. saginata, T. solium, H. nana, H. diminuta, D. caninum.

Larval cestodes of man are:
1- Sparganum (plerocercoid) larva of D. mansoni and D. proliferum.
2- Cysticercus cellulosae of T. solium.
3- Hydatid cyst of E. Granulosus and E. multilocularis.
4- Coenurus cyst of M. multiceps.

Man can act as definitive and intermediate host for the same cestode in:
T. solium and H. nana.

Hydatid cyst of Echinococcus Granulosus:
1- Thy fully developed cyst is typically unilocular, spherical in shape and filled with fluid.
2- It reaches diameter if 10 cm or more (this takes many years).
3- In humans, 80 – 90 % of hydatid cysts are found in liver or lung, others are found in brain, bones and kidneys.
4- The cyst wall is formed of three layers from inside to outside:
   a. Cellular or germinal layer, capable of division.
   b. Elastic non cellular laminated layer.
   c. Host produced fibrous layer to prevent further growth of the cyst.
5- The cyst contains:
   a. Individual scolices (microscopic, 100 – 1000).
   b. Daughter cysts similar to the mother cyst.
   c. Brood capsules which are sacs enclosing a number of scolices. Scolices, daughter cysts and brood capsules may remain attached to the wall of the of the mother cyst or detach and fall into the cavity of the mother cyst (called hydatid sand).
6- Exogenous daughter cyst occurs as a result of herniation of germinal layer to the outside.
7- Sometimes, the germinal layer of the mother & daughter cysts and brood capsules fail to give scolices, thus we get sterile cyst.

Parasite (Disease)	Multiceps multiceps (Coenurosis)	Dipylidium Caninum (Dipylidiasis)
Geographical Distribution	Cosmopolitan	Cosmopolitan
Definitive Host & Habitat	Small intestine of dogs and canines.	S.I. of dogs, cats & man occasionally.
Diagnostic Stage	See diagnosis.	Gravid segments or egg capsules.
Intermediate Host	Sheep, goats and occasionally man.	Flea larvae of dogs and cats.
Infective Stage & Mode Of Infection	Ingestion of eggs with infected food, drink or hands.	Ingestion of infected fleas.
Life Cycle	➢It develops the same way as hydatid cyst. ➢The coenurus cyst develops chiefly in the brain and spinal cord.	Adult worms on small intestine of D.H. → eggs (oncospheres) → faeces → ingestion by I.H. → cysticercoids → ingestion of I.H. by D.H. → small intestine → Adults
Pathogenesis and Clinical Picture	Symptoms of increased intra cranial tension.	1- Usually asymptomatic. 2- Abdominal pain and diarrhea may occur.
Diagnosis	As a space-occupying lesion in the brain or spinal cord but confirmed as coenurus cyst after surgical removal.	By finding gravid segments or egg capsule in stool.
Treatment	Surgical removal.	As in taeniasis.
Prevention & control	As hydatid disease.	----------

Class: Nemathelminths
Subclass: Nematoda

General characters:
1- Elongated and cylindrical → round in cross section.
2- Unsegmented with body cavity.
3- Separate sexes: posterior end is curved in most of males and straight in females.
4- Body wall consists of 3 layers:
   a. Outer laminated (cuticle).
   b. Sub-cuticle (hypodermis).
   c. Muscular.
5- Digestive system: simple tube extending from mouth to anus and formed of: mouth, esophagus and intestine.
6- Genital system:
   a. Male system: one genital set.
   b. Female system: two genital sets except in Trichuris and Trichinella. Female worm may either:
   - Give birth to larvae (larviparous).
   - Lay eggs (oviparous) either immature or mature.

Eggs of nematoda	Immature	One cell e.g. Ascaris, Trichuris
		4 cells e.g. Ancylostoma, Capillaria
		16 – 32 cells e.g. Trichostrongylus
	Mature	e.g. Enterobius, Strongyloides

Tissue nematodes characters:
1- The adult worms live in the tissues of man (extraintestinal).
2- The oesophagus is filariform (cylindrical).
3- The female is larviparous (laying larvae).
4- An arthropod vector (Intermediate host) is required for transmission.

Modes of infection of nematodes:
1- Ingestion either by:
   a. Ingestion of eggs:
      i. Eggs pass infective e.g. Enterobius.
      ii. Eggs become infective after a period of maturation outside e.g. Ascaris and Trichuris.
   b. Ingestion of larvae:
      i. In vegetables or water e.g. Trichostrongylus.
      ii. In pig muscle e.g. Trichinella.
      iii. In fish e.g. Capillaria.
      iv. In Cyclops e.g. Dracunculus.
2- Penetration of skin:
   a. Larvae penetrate the skin e.g. Ancylostoma, Strongyloides.
   b. Through bite of blood sucking insects e.g. Filaria.

Protective mechanisms of nematodes:
1- Intestinal nematodes resist the action of digestive juices by their cuticle and lytic enzymes secreted by the worm.
2- They maintain their position by:
   a. Oral attachment to the mucosa by teeth or plates (Hookworms).
   b. Partial penetration of the mucosa (Trichuris and Trichostrongylus).
   c. Complete penetration of the mucosa (Strongyloides, Trichinella and Capillaria).
   d. Retention of the folds of mucosa and pressure against it (Ascaris, Enterobius).

Parasite (Disease)	Diphyllobothrium latum (Diphyllobothriasis)	Diphyllobothrium mansoni & proliferum (Sparganosis)	Taenia saginata (Taeniasis Saginata)	Taenia solium (Taeniasis Solium)	Cysticercosis
Geographical Distribution	Lake regions, not in Egypt. Can be imported in fish.	Far east, USA, East Africa.	Cosmopolitan especially in cattle raising countries.	Cosmopolitan especially in pork raising countries.	
Definitive Host & Habitat	Small intestine of man.	Small intestine of cats and dogs.	Small intestine of man only.	Small intestine of man.	Tissues of man.
Reservoir Host	Fish eating animals: dog & cat	----------	----------	----------	----------
Diagnostic Stage	Immature egg stage: Size: 70 × 50 um. Color: yellowish brown. Shape: oval, operculated & thick shelled. Content: immature ovum.	Cannot be settled except after surgical removal and identification of plerocercoid larva in removed tissue.	Immature egg stage: Size: 30-40 um in diameter. Color: yellowish brown. Shape: spherical with radially striated shell. Content: hexacanth oncosphere. Ziehl Nielsen stain: T. saginata stained well but T. solium not		----------
Intermediate Host	1st: Cyclops (water flea). 2nd: fresh water fish: Salmon.	1st: Cyclops. 2nd: frogs, snakes, mammals, birds, or man (blind end).	Cattle	Pig	----------
Infective Stage & Mode Of Infection	Plerocercoid larvae through ingestion of undercooked or under salted contaminated (infected salmon) fish.	Plerocercoid larvae through: **1-**Ingestion of undercooked flesh of 2nd I.H. **2-**Drinking water containing infected Cyclops. **3-**Applying the flesh of 2nd I.H. as foment or poultice to inflamed tissue as skin or eye.	Cysticercus bovis through ingestion of undercooked infected beef.	Cysticercus cellulosae through ingestion of undercooked infected pork.	Ingestion of eggs by: **1-**Heteroinfection: through infected food or water. **2-**External autoinfection: hand to mouth infection in infected patient. **3-**Internal autoinfection: some detached segments of the worm ascend against peristaltic movement of intestine then descend again where they hatch and cause cysticercosis.
Life Cycle	Adult worms in small intestine→ Eggs→ Faeces→ Fresh water → Coracidium→ 1st I.H.→ Procercoid larva → 2nd I.H. eat 1st I.H. → penetrate intestinal wall → tissues & muscles → plerocercoid larva or sparganum → Ingestion by D.H. → Small intestine → Maturation (6 weeks)→ Eggs	Adult worms in small intestine of dogs & cats → 1st I.H.→ Procercoid larva → 2nd I.H (occasionally man) → plerocercoid larva or sparganum → any tissue (man is a blind end of the cycle because he is not eaten by other animals)	Adult worms in small intestine→ gravid segment detach **singly** → out with faeces or by creeping→ perianal region → Eggs→ grass →ingestion by cattle→ penetrate intestinal wall → blood → muscles → Cysticercus bovis → Ingestion by D.H. → Small intestine → Maturation (3 months) → Eggs	Adult worms in small intestine→ gravid segment detach in **chains** → through anus →perianal region → Eggs → grass →ingestion by pigs → penetrate intestinal wall → tissues & muscles → Cysticercus cellulosae → Ingestion by D.H. → Small intestine → Maturation → Eggs	----------
Pathogenesis and Clinical Picture	**1-**May be asymptomatic. **2-**Intestinal disturbance: colic, hunger pain, nausea, vomiting, diarrhea and loss of appetite. **3-**Neurological manifestations: headache, insomnia or convulsions caused be absorbed toxins. **4-**Large no. may produce intestinal obstruction. **5-**Pernicious anemia (macrocytic hyperchromic): **a.**Some toxins. **b.**Vit. B_{12} deficiency because the parasite competes for it.	Depend on the tissue invaded: **1-**Skin: inflammatory tender swellings. **2-**Eye: painful edematous conjunctivitis and ptosis. **3-**Degenerated larvae: cause inflammation and necrosis but no fibrosis. **4-**Patient may suffer from: urticaria, edema, fever, pain and eosinophilia.	**1-**Intestinal disturbance. **2-**Neurological manifestations. **3-**Intestinal obstruction. **4-**Loss of weight & hunger pains as the parasite consumes much of patient's food. **5-**Appendicitis or cholangitis caused by stray segments of the worm. **6-**Migrating segments creeping out of the anus cause irritation, itching and worry of the patient.	**1-**Intestinal disturbance, Neurological manifestations, Intestinal obstruction & loss of weight. **2-**If man ingests the eggs, the larval stage develops in extra-intestinal tissues → cysticercosis. This condition occurs with T. solium only which makes it more dangerous.	**1-**Sites: brain, subcutaneous tissue, eye, heart or any other tissue. **2-**Cysts produce inflammatory reactions which usually end by fibrosis and calcification. **3-**Muscle pain, fever and eosinophilia. **4-**Cysts in subcutaneous tissue are easily palpated (lipoma). In the eye may lead to visual disturbances. In neuro-cysticercosis leads to variable neurological disorders.
Diagnosis	**1-**Detection of eggs and segments in Faeces. **2-**Blood picture shows anemia.	Cannot be settled except after surgical removal and identification of plerocercoid larva in removed tissue.	**1-**Detection of eggs or segments in Faeces. **2-**Recovery of eggs from perianal region by swab. **3-**Searching for gravid segment in Faeces. If not found, give a saline purge.	**1-**Detection of eggs in Faeces & differentiation by Ziehl Nielsen stain. **2-**Detection of gravid segments in Faeces.	**1-**Intestinal infections. **2-**Biopsy from a nodule in skin or muscles. **3-**X-ray to visualize calcified lesions. **4-**CT, MRI, ultrasonic or oph-thalmoscopic examinations. **5-**IHA, ELISA, eosinophilia & intra-dermal tests.
Treatment	**1-**Praziquantel. **OR** **2-**Niclosamide (Yomesan). **3-**Supportive treatment: Vit. B_{12} given parenterally.	Surgical removal (difficult in sparganum proliferum due to its proliferation and spread to other tissue).	**1-**Praziquantel. **OR** **2-**Niclosamide.	**1-**Praziquantel and Niclosamide. **2-**A saline purge is given 1-2 hours later to wash the eggs to prevent cysticercosis. **3-**Quinacrine hydrochloride (atebrine) for expulsion of the intact parasite.	**1-**Surgical treatment. **2-**Praziquantel. **3-**Albendazole. **4-**Simultaneous administration of steroids to relieve intense inflammatory reactions. **5-**Vit D & calcium to help calcification.
Prevention & Control	**1-**Sanitary disposal of human excreta. **2-**Proper cooking of fish. **3-**Treatment of infected patients. **4-**Periodic de-worming of reservoir hosts. **5-**Health education.	**1-**Water should be boiled or filtered. **2-**Thorough cooking of flesh of I.H. **3-**Avoiding fomentation with the flesh of I.H.	**1-**Treatment of infected men. **2-**Preventing contamination of soil by human Faeces. **3-**Protection of I.H. by preventing them from grazing in infected areas. **4-**Proper inspection of slaughtered cattle. Infected carcasses must be condemned. **5-**Proper cooking or deep freezing of meat.	Same as T. saginata but mainly directed towards pigs.	**1-**Sanitary disposal of human excreta. **2-**Pure water supply. **3-**Proper washing of vegetables. **4-**Treatment of infected patients. **5-**Health education.

Parasite (Disease)	Echinococcus granulosus (Hydatidosis, Hydatid disease)	Echinococcus multilocularis	Hymenolepis nana (Hymenolepiasis)	Hymenolepis diminuta (Hymenolepiasis diminuta)
Geographical Distribution	Cosmopolitan, in sheep raising countries.	In cold areas	Cosmopolitan, in warm areas, in Egypt too.	Cosmopolitan
Definitive Host & Habitat	Small intestine if dogs & canines <u>but not man</u>.	Small intestine of foxes, wolves and cats.	Small intestine (S.I.) of man.	S.I. of rats, mice & occasionally man
Diagnostic Stage	Hydatid cyst	The alveolar cyst	Egg stage: <u>Size</u>: 30-50 um diameter <u>Color</u>: Translucent. <u>Shape</u>: spherical with two coverings. <u>Content</u>: mature hexacanth oncosphere.	
Intermediate Host	Herbivorous animals.	----------	Flea larvae or grain beetles.	
Infective Stage & Mode Of Infection	Egg stage through: - Hand to mouth from fur of infected animals. - Food or drink infected by animal faeces.		Egg stage or cysticercoid larva, by: **1.** Ingestion of contaminated food and water. **2.** Autoinfection (hand to mouth).	Ingestion of insect vector.
Life cycle	<u>Adults</u> in S.I. of D.H.→ <u>eggs (oncospheres)</u> → faeces → grass → ingestion by I.H. → penetrate S.I. → pass to blood by lymphatics or venules → various parts of body → vesiculation → grow slowly → <u>Hydatid cyst</u> (take several years & may be single or multiple).	----------	**<u>Direct cycle</u>**: <u>Adults</u> in S.I. of D.H. → <u>eggs</u> → faeces → ingestion by man → penetrate S.I. mucosa → <u>cysticercoid larva</u> → after 1 weak → return to the lumen → <u>adult stage</u>. **<u>Indirect cycle</u>**: <u>Adults</u> in S.I. of D.H. → <u>eggs</u> → faeces → eaten by I.H. → <u>cysticercoid larva</u> → ingestion of I.H. accidentally by man → S.I. → <u>adult stage</u>. (*H. diminuta shows this cycle only*).	
Pathogenesis & Clinical Picture	**<u>Hepatic cyst (66%):</u>** Usually in the right lobe extending towards the abdominal cavity: **1-**May cause no symptoms until it expands. **2-**Obstructive jaundice. **3-**Rupture of the cyst leads to: **a.** 2ry new cysts with hydatid sand or bits of germinal layer. **b.** Rupture into bile ducts leads to intermittent jaundice, fever and eosinophilia. **c.** Allergic manifestations up to anaphylactic shock in case of entrance of hydatid material to blood stream. **<u>Pulmonary cyst (22%):</u>** **1-**Early symptoms include hemoptysis, transient thoracic pain and shortness of breath. **2-**In majority of cases, the cyst transfer into chronic abscess (if rupture is incomplete) and patient complains of sudden attack of cough with sputum contains frothy blood, mucous & hydatid material. **<u>Brain cysts (1%)</u>** Large cyst→ ↑intracranial tension up to epilepsy **<u>Renal cysts (3%):</u>** Intermittent haematuria. Hydatid sand may be present in urine. **<u>Osseous cysts (2%):</u>** It has no fibrous nor laminated layers, but only germinal layer which develops in bone marrow cavity then extends to osseous tissue leading to: **1-**Erosion of large area of bone. **2-**Destruction of trabeculae. **3-**Spontaneous fracture.	**<u>The alveolar cyst</u>** It is a porous spongy gelatinous mass formed of small irregular cavities that are lined by germinal layer with a very thin or no laminated layer with fibrous tissue strands in-between the cavities: • It has irregular outline which is not defined from the surrounding tissues. • It behaves like a malignant tumor i.e. degeneration and calcification in the center and spreading at the periphery. It gives metastasis through blood or lymph. • Its commonest site is in the liver (90-100%). • In human, the cyst is usually sterile (no scolices in the fluid medium of the cyst).	**1-**<u>Light infection</u>: asymptomatic **2-**<u>Heavy infection</u>: • Abdominal pain • Appetite loss • Diarrhea or vomiting • Nervous manifestations as dizziness, insomnia and convulsions due to absorption of toxic byproducts of the worm.	**1-**Usually asymptomatic. **2-**Mild GIT disturbances occasionally.
Diagnosis	**<u>Clinically</u>** by detection of slowly growing cystic tumor & history of contact with dogs. **<u>Ultrasonography & CT</u>** detect un-calcified cysts & of value in follow up of treated cases. **<u>X-ray imaging</u>** especially in pulmonary cysts and calcified cysts: **1-**Round solitary or multiple sharply contoured cysts of 1 – 15 cm in diameter. **2-**Internal daughters give a car wheel shape **3-**Thin crescent or ring shape calcification. **<u>Serological tests</u>**: IHA, ELISA. **<u>Aspiration cytology</u>: risky** **<u>Molecular diagnosis</u>**: DNA analysis & PCR. **<u>Intradermal test of Casoni</u>**: was used but may give false results in 18% of cases. Now it is not preferred because it may give allergic reactions.	----------	Detection of eggs in faeces.	
Treatment	**1-**Surgical removal. **2-**PAIR technique. **3-**Medical ttt: **i-**Albendazole **ii-**Praziquantel	Surgical removal	**1.** Praziquantel **2.** Treat all members of the family at the same time (mass ttt).	
Prevention & Control	**1-**Proper disposal of infected viscera. **2-**Elimination of dogs. **3-**Periodic examination of pet dogs and treatment of infected ones by Praziquantel. **4-**Avoid dogs and prevent children from playing with them. **5-**Protection of food and drink from infected dogs.	----------	**1-**Personal hygiene. **2-**Mass treatment. **3-**Avoid infected food and drink. **4-**Elimination of arthropods & rodent control. **5-**Environmental sanitation.	Rodent control.

Parasite (Disease)	**Ascaris Lumbricoides (Ascariasis)**	**Trichuris Trichiura (Trichuriasis)**	**Enterobius vermicularis (Enterobiasis)**
Geographical Distribution	Cosmopolitan, common in warm areas with bad sanitation, in Egypt too (especially in children).		Cosmopolitan, common in temperate & cold climates.
Definitive Host & Habitat	Man, live free in the lumen of the small intestine.	Man, in the caecum and adjacent parts.	Man (only), in the caecum and adjacent parts of small & large intestine and appendix.
Diagnostic Stage	Egg stage. Eggs are the most resistant. They can survive for months and years in soil. See the details in the next page.	Immature egg stage: Size: 50 × 25 um. Color: brownish. Shape: barrel shaped, thick shelled with a mucoid plug at each pole. Content: immature ovum (1 cell stage).	Adult stage or egg stage, egg characters: Size: 50 × 25 um. Color: translucent. Shape: plano-convex, has 2 layers covered by outer sticky albuminous layer. Content: larvae (infective in few hours).
Infective Stage & Mode Of Infection	2nd stage rhabditiform larvae through ingestion of embryonated eggs.	Rhabditiform larvae by ingestion of embryonated eggs through contaminated water, vegetable or hands.	Infective eggs containing larvae, through: • Autoinfection (hand to mouth). • Contaminated food or drink. • Handling contaminated linen, clothing or articles. • Air-born infection. • Retro-infection.
Life Cycle	Adult→eggs→faeces→soil → 1st rhabditiform larva →1st moult → 2nd rhabditiform larva → ingestion → penetrate S.I.→ venous blood → lung → enter alveoli → 2nd & 3rd moult → ascend in the respiratory tracts → swallowed →S.I. →4th moult → adult	Adults in caecum → eggs → soil → shade & moisture → Rhabditiform larvae → ingestion → lower part of S.I. → caecum → moult 4 times → adult	Adults → female migrate towards the anal opening → lay sticky eggs → perianal area → infection by any mode → larvae → hatch in S.I. → moult twice → adult
Pathogenesis	Tissue damage due to: **1-**Large size of Ascaris (largest intestinal nematode). **2-**Adults do not attach to intestinal wall, and may go ectopic places. **3-**Toxic products stimulate immune response.	The embedded anterior parts of the worms cause inflammation and irritation of the mucosa with hemorrhage. Secondary infection results in sub-mucosal abscesses & ulcers.	
Clinical Picture	**1-**Usual infection (10-20) worms pass unnoticed. **2-**During larval migration: **a.** Pneumonitis: fever, cough, dyspnea & eosinophilia. **b.** Allergic reactions asthmatic attacks & edema of lips **c.** Sputum examination reveals streaks of blood, eosinophils & may be larvae. **d.** Loeffler's syndrome: x-ray shows scattered mottling. **e.** Ectopic lesions. **3-**In the intestine: **a.** Abdominal pain, nausea, vomiting, colic, distention or dyspepsia due to production of anti-enzymes that interfere with digestion → malnutrition. **b.** Changes in the bowel movements (diarrhea or constipation). **4-**Complications: **a.** Traumatic effects due to irritation of the worms which may go to: • Bile duct → obstructive jaundice. • Liver → abscesses. • Appendix → appendicitis. • Ampulla of Vater → acute hemorrhagic pancreatitis • Peritoneum → Peritonitis. • Stomach → vomiting or escape through nares • Trachea (rare) → suffocation. **b.** Toxic effects by toxins that may produce oedema, asthma, insomnia, irritability & convulsions. **c.** Larvae in ectopic sites give the picture of visceral larva migrans.	***Mild infection:*** Usually asymptomatic. ***Moderate infection:*** • Frequent small blood-streaked stool (bloody diarrhea). • Pain and tenderness in the lower abdomen. • Nausea, vomiting & loss of weight. ***Heavy infection:*** • Dysentery: the worms are distributed throughout the colon & rectum leading to oedemtous hyperemic fragile mucosa. • Rectal prolapse: due to chronic straining due to dysentery leads to loss of anal tone & prolapse. • Anemia: due to suction & bleeding causing microcytic hypochromic anemia. Toxic by-products may cause macrocytic hyperchromic anemia (Trichocephalic anemia). • Rarely perforation: lead to peritonitis. • May invade appendix: → appendicitis. • Eosinophilia: is persistent.	**1-**Pruritus ani: itching in the perianal area especially at night. **2-**Nervous irritability, hyperactivity, insomnia and 2ry enuresis. **3-**Female migration to ectopic sites stimulate granuloma formation, through migration to: **a.** Vagina → vulvo-vaginitis, they may migrate to uterus or fallopian tubes. **b.** Urinary tract → infection & enuresis. **c.** Appendix → appendicitis. **d.** Intestine → diarrhea & abdominal pain. **e.** Peritoneal cavity (through uterine tubes) → pelvic peritonitis.
Diagnosis	**1-**Clinically: Transient cough & dyspnea which disappear after 1-2 weeks followed by vague abdominal manifestations. **2-**Laboratory investigations (findings): **a.** Eggs in faeces. **b.** Adults in faeces, vomits, or intestinal obstruct. **c.** Larvae in sputum with blood & eosinophils. **d.** Eosinophilia: 20 % during migration then regresses to 7 %. **3-**Radiologically: **a.** Plain x-ray: adults appear as gas-filled loops. **b.** Barium meal: filling defects represent adults.	**1-** Finding eggs in stool. **2-** Rectal examination by proctoscopy: hyperemic edematous mucosa with hanging worms. **3-** Air-contrast barium enemas: linear translucent adults in contrast to barium-coated bowel mucosa. **4-** Blood-test: - Eosinophilia (5 – 15 %). - Anemia.	**Clinically:** Infection is suspected in children with pruritus at night. **Laboratory:** • Adult worms may be seen in stool or anal area. • Eggs are rarely found in stool (about 5% only). • Swabbing of anal or perianal area by: ○ N.I.H. swab (National Institute of health): the peri-anal area is swabbed in the morning before defecation or bathing with a cellophane paper folded and tied to tip of a glass rod and inserted in a test tube. The cellophane is stretched in a slide and examined microscopically for eggs. ○ Scotch adhesive tape swab: Scotch tape with sticky side outwards is pressed against perianal area then spread on a slide with sticky side downwards and examined microscopically.
Treatment	**1-** Albendazole **OR** Mebendazole. **2-** In mixed infections, it is advisable to treat Ascaris first (to avoid worm irritation → ectopic lesions). **3-** Surgical treatment of complications.	**1.** Albendazole **OR** Mebendazole. **2.** Repeated course may be necessary. We have to give anti-diarrheal drug before ttt.	**1.** Albendazole **OR** Mebendazole: it should be repeated after 2 weeks. **2.** Local application of white oxide of mercury around the anus to relief the itching & kill the out coming worms.
Prevention & Control	**1-** Mass treatment. **2-** Washing hands before meals. **3-** Sanitary disposal of human faeces. **4-** Proper washing of fruits and vegetables eaten raw. **5-** Night soil should not be used as fertilized unless treated by chemicals.		**1-** Mass treatment. **2-** Personal hygiene. **3-** Toilet seats disinfected frequently. **4-** Food protection. **5-** Infected children should use tight trousers at night to prevent auto-infection.

Egg stage in Ascaris Lumbricoides

Egg type	Fertilized egg	Unfertilized egg	Decorticated egg
Size	60×45 um	90×40 um	-------------
Shape	Oval, thick smooth layer covered by mamillated albuminous coat.	Longer and narrow with ill-defined mamillations.	Fertilized egg but lacking the mamillations.
Color	Brownish		
content	Immature(one cell stage)		

Life cycle of Strongyloides stercoralis:

Direct cycle (similar to hookworm):

Rhabditiform larvae → soil → moult → infective filariform larvae → penetrate the skin → venous circulation → lungs → penetrate the alveoli → migrate through the trachea → swallowed → small intestine →2 moults → adults

Indirect cycle (if the soil condition is optimal):

Rhabditiform larvae → soil → four moults within 2 days →adult (free-living) → mature ova → Rhabditiform larvae (free-living) as long as the conditions are suitable. If the condition becomes unfavorable, the rhabditiform larvae become infective filariform larvae.

Autoinfection:

- When a person suffers from constipation, rhabditiform larvae have enough time to moult into infective filariform larvae. Then they penetrate the mucosa of large intestine then complete the cycle (internal autoinfection).
- Also, the infective filariform larvae can penetrate the perianal skin after coming out from the anus and then complete the cycle (external autoinfection).

Parasite (Disease)	Hookworm - Ancylostoma duodenale (Ancylostomiasis)	Strongyloides stercoralis (Strongyloidiasis)	Trichostrongylus colubriformis (Trichostrongyliasis)
Geographical Distribution	Mediterranean, North Africa, South America, India and China.	Cosmopolitan, more in tropical and subtropical countries.	Cosmopolitan, especially in agricultural areas.
Definitive Host & Habitat	Small intestine S.I. (jejunum) of man only.	Man, in the duodenum and upper jejunum, but in heavy infection may involve the whole intestines	Upper part of S.I. of herbivorous animals and occasionally man (may invade biliary passages).
Diagnostic Stage	Immature egg stage: Size: 40 × 60 um. Color: Translucent. Shape: oval with rounded poles & thin shelled. Content: immature ovum with 4 cell stage.	Filariform larvae.	Immature egg stage: Size: 80 × 40 um. Color: Translucent. Shape: oval, thin shelled with one round pole & the other pointed. Content: immature (morula stage, 16-32 cells).
Infective Stage & Mode Of Infection	Filariform larva through penetration of the skin results from handling soil without gloves or shoes	Filariform larvae through penetration of skin or the mucosa of the intestine.	Ensheathed filariform larvae through ingestion with green vegetables and water.
Life Cycle	Adults in S.I.→eggs →faeces→soil→ 1st stage rahbditiform larva → 1st moult → 2nd stage rahbditiform larva → 2nd moult → infective filariform larva → penetrate the skin → venules or lymphatics → lungs → 3rd moult → penetrate the alveoli → migrate through the trachea → swallowed→small intestine→4th moult→adult	Adults in S.I. → eggs → inside the mucosa of intestinal villi → rhabditiform larvae → lumen → faeces → has 3 types of life cycle: **1-** Direct cycle (similar to hookworm). **2-** Indirect cycle. **3-** Autoinfection. See the details above this table.	Adults in S.I. → eggs → faeces → soil → rhabditiform larvae → moult 2 times within 4 – 5 days → ensheathed filariform larvae → ingestion → S.I. → another moult → penetrate the villi → remain for 4 days → back to lumen → Adults
Pathogenesis & Clinical Picture	***Skin lesions***: itching, erythema, vesiculation and pustulation at the site of penetration due to 2ry bacterial infection (ground itch or hookworm dermatitis). ***Pulmonary lesion***: asthmatic bronchitis, minute hemorrhage, verminous pneumonitis, rise to: fever, cough, dyspnea, hemoptysis & eosinophilia (up to 70%) after 2-3 weeks (Loeffler's syndrome) *These 2 stages are seen in individuals who receive a primary infection.* ***Intestinal lesion***: - Hemorrhage (0.3 cc blood/day) results from attachment of the parasite to the mucosa by its cutting teeth. The worms leave the oozing site & attach to other site and so causing minute ulcers - Hypochromic microcytic anemia results from chronic blood loss & depletion of iron stores. It results in pallor, fatigue, dyspnea & tachycardia - Subcutaneous edema due to hypo-proteinaemia - GIT: nausea, vomiting & diarrhea due to mucosal ulcerations. Melaena & occult blood in stool may occur. - Pica i.e. habitual ingestion of non-food substances as soil. - Retardation of physical and mental development	***Skin lesions***: dermatitis & itching. Larvae may remain in the skin producing cutaneous larva migrans which usually seen in patients who develop external autoinfection. The lesion starts at the perianal region and extends as linear eruption across the buttocks, thigh & back at a fast rate (5 – 10 cm/hour) referred to as larva currens. ***Lung lesions***: minute hemorrhage & pneumonitis as hookworms. ***Intestine lesions***: • Burning epigastric pain with tenderness (duodenitis). • Nausea and vomiting, diarrhea alternates with constipation. • Long-standing heavy infection results in weight loss, chronic dysentery, mal-absorption and steatorrhoea. ***Disseminated Strongyloidiasis***: Occasionally some larvae pass through the pulmonary barrier to the left side of the heart to reach various organs of the body. In-patient with impaired immunity, the parasite produces massive number of larvae, which penetrate to extra intestinal organs and could be fatal. So it is considered as an opportunistic parasite.	❖ Infection is usually light producing no symptoms. ❖ Heavy infections may cause anemia or signs of cholecystitis.
Diagnosis	**1-**Clinical: above. **2-**Laboratory: **a.** Stool examination for eggs. **b.** Determination of anemia. **c.** Testing for occult blood in stool.	**1-** Examination of faeces or duodenal contents for larvae either by: **a.** Direct or concentration methods which reveal the motile larvae in fresh specimen. **b.** Culture for 48 hours gives free living adult worms. **2-** Examination of sputum for larvae. **3-** Eosinophilia. **4-** Serological test as ELISA.	**1-** Finding eggs in stool or duodenal aspirate. **2-** Stool culture may give larvae.
Treatment	**1-**Albendazole **OR** Mebendazole (vermox). **2-**Iron supplement and protein rich diet.	**1-** Thiabendazole. **OR** **2-** Ivermectin.	Thiabendazole (Mintezol)
Prevention & Control	**1-**Sanitary disposal of human excreta. **2-**Mass treatment. **3-**Disinfection of human excreta used as fertilizers. **4-**Wearing shoes and gloves. **5-**Killing the filariform larvae using soil larvicides. **6-**Health education: avoid being barefooted and defecation on the ground and use of latrines (toilets)		**1-** Treatment of infected animals or patients. **2-** Proper washing of green raw vegetables and pure water supply.

Name Parasite (Disease)	Larva Migrans: Cutaneous larva migrans (CLM)	Larva Migrans: Visceral larva migrans (VLM)	Capillaria philippinensis (Intestinal Capillariasis)	Trichinella spiralis (Trichinosis)
Geographical Distribution	-------------	-------------	Philippines & Thailand, some cases detected in Egypt.	Cosmopolitan, especially in pork-eating countries.
Definitive Host & Habitat	Not the man, so they cannot complete their cycle, instead they migrate under the skin.	Dogs & cats, not man so they invade man viscera & cannot complete the cycle.	Fish eating birds and occasionally man, embedded in the mucosa of jejunum & ileum.	S.I. of man, pigs and rodents (rats). <u>They act as intermediate hosts (I.H.) too.</u>
Diagnostic Stage	-------------	-------------	-------------	-------------
Infective Stage & Mode Of Infection	Filariform larvae through penetration of skin.	Infective egg through ingestion of contaminated food, drink or hands.	Larval stage, through eating raw or poorly cooked fish and internal auto-infection.	Encysted larvae, through ingestion of improperly cooked infected pork.
Life Cycle	-------------	-------------	<u>Adult female</u> → <u>eggs</u> → fresh water → <u>embryonated eggs</u> → I.H. (fish) → S.I. → <u>larvae</u> → ingestion of I.H. by D.H. → S.I. of D.H. → S.I. of D.H. → hatch → <u>adult</u> **In autoinfection**: <u>Female</u> → <u>eggs & larvae</u>→invade the mucosa→<u>adult</u>	<u>Larvae</u> → ingestion by pigs → its muscles → ingestion by man→ S.I.→ 3 moults→ <u>adults</u> → <u>female</u> → <u>eggs</u> in the mucosa→ <u>larvae</u>→ blood→ all tissues especially striated muscles→ coiled → encyst → <u>encysted larvae</u>
Pathogenesis & Clinical Picture	**1.** The lesion starts as a red itchy papule at the site of entry followed by a slightly elevated erythematous serpiginous tunnel 1-2 mm in diameter with itching and 2ry infection. **2.** The lesion advances at a rate of 1-2 cm/day for several weeks or months till the larvae die. This commonly seen in the skin of hands, feet, back of buttocks.	**1.** The rhabditiform larva hatch in the S.I. & penetrate the wall → circulation → viscera (liver mainly) → wander for weeks or months or become dormant causing eosinophilic granulomatous lesion. **2.** The characteristic granuloma consists of a gray elevated circumscribed area about 4 mm in diameter. It consists of eosinophils, lymphocytes & foreign body giant cells surrounding the larva. **3.** Symptoms depend on location of larvae & the patient's allergic response: - Asymptomatic with persistent eosinophilia. - The usual picture is: ○ Child 1- 4 years old. With history of contact with soil, dogs & cats. ○ Marked persistent eosinophilia (20 – 80 %). ○ Enlarged tender liver. ○ Pneumonitis & pulmonary infiltration may be seen in x-ray. ○ Visual or neurological disturbances. ○ Marked increased blood γ-globulins.	**1.** Pathogenesis depends on the presence of parasitic stages in the mucosa → chronic inflammation reactions → atrophy → mal-absorption of fats, sugars, proteins and electrolytes. **2.** Abdominal pain, chronic diarrhea, vomiting, low-grade fever, dehydration, loss of weight & oedema of lower limbs (due to hypo-proteinaemia). **3.** Death may occur due to severe electrolyte imbalance or due to superimposed bacterial infection.	**<u>Intestinal stage (1st week)</u>**: Gastro-enteritis → nausea, vomiting, abdominal cramps and diarrhea simulating ingestion of infected pork. **<u>Stage of larval migration (2nd week):</u>** Fever, oedema of eye lids, myositis & weakness of invaded muscles. There may be shallow rapid breaking eosinophilia 20–50%. **<u>Stage of encapsulation (3rd week)</u>**: Fever recovers slowly, muscle pain is persistent. Death may occur from myocarditis, pneumonia or encephalitis in case of severe infections.
Diagnosis	**1.** Clinically depends on the advancing serpiginous tunnels & history of contact of skin with soil. **2.** Suspect migration of larvae in the tissues if there is high eosinophilia. The larva is always ahead of its track.	**1.** Clinically: a young child, with chronic eosinophilia, exposed to ascarid-infected pets, eating soil, hepatomegaly or chronic pulmonary disease is suggestive. **2.** Laboratory diagnosis: - Laparoscopy & biopsy of liver nodules under vision is better than needle biopsy. - Hyper-γ-globulins: ↑IgG, IgM, IgE. - Eosinophilia (20 – 80 %). - Elevated anti-A & anti-B iso-haemo-agglutinin titre due to cross reactivity with larval antigen. - Serological tests: IHA, IFA, ELISA.	**1.** Clinical examination: above. **2.** Stool analysis: all stages of the parasite are detected in watery stool with a lot of Charcot Leyden crystals. **3.** Laboratory investigations: - Low serum Na, K, Ca. - Low serum proteins (especially albumin).	**<u>Clinically</u>**: A history of eating pork with fever, eosinophilia, facial oedema & myositis is suggestive. **<u>Laboratory diagnosis</u>**: **1-** Muscle biopsy: examined for larvae. **2-** Eosinophilia: 10-90% in the 3rd to 4th week. **3-** Intradermal test. **4-** Serological tests: as IFAT & ELISA. **5-** X-ray showing calcified cysts.
Treatment	**1.** Albendazole. **2.** Thiabendazole ointment. **OR** **3.** Thiabendazole (Mintezol). **4.** Antibiotics for 2ry infection. **5.** Anti-histaminics.	**1.** Thiabendazole (Mintezol). **2.** Corticosteroids in severe cases.	**1.** <u>Specific</u>: Mebendazole **OR** Albendazole. **2.** <u>Supportive ttt</u>: fluids, electrolytes, high protein diet and vitamins.	**1-** Mebendazole (vermox) **OR** Thiabendazole. **2-** Corticosteroids. **3-** Symptomatic treatment: for fever, headache & muscle pain.
Prevention & Control	**1.** Avoid skin contact with soil polluted with dog or cat faeces. **2.** Regular examination & treatment of pet animals and elimination of stray dogs & cats.	**1-** Dogs & puppies should be kept away from children. **2-** Pets should be de-wormed regularly & elimination of stray ones. **3-** Avoid contamination of food, drink & hands by excreta of dogs & cats and soil.	It impossible to control birds, but in human it is necessary to: **1-** Detect & treat cases. **2-** Prevent contamination of Lagoons by sanitary disposal of human excreta. **3-** Warning people of the danger of eating raw fish.	**1.** Destruction of rats & proper breeding of pigs. **2.** Heat ttt of garbage fed to swine. **3.** Avoidance of eating pork. **4.** Meat inspection of slaughter houses (Trichinoscope). **5.** Destruction of larvae by proper cooking & freezing (at -15° for 20 days or quick at -37°). **6.** Pork roasts cooked in micro-wave ovens does not kill larvae.

Other causes of cutaneous larva migrans:

1-Human and non-human strains of Strongyloides (larva currens; fast moving).

2-Cutaneous myiasis caused by larvae of flies as Gastrophilus and Hypoderma.

N.B. In case of heavy infection by VLC, some larvae of Ascaris, Ancylostoma & Strongyloides, during their cycle, pass from the lungs to the left side of the heart to the systemic circulation and settle in different organs producing visceral larva migrans.

Name Parasite (Disease)	Dracunculus medinensis (Dracunculiasis, Dracontiasis)	Wuchereria bancrofti (Bancroftian filariasis, Elephantiasis)
Geographical Distribution	In areas where people depend on wells for water supply. Most cases are in Africa (Sudan, Mali & Ghana)	In tropical and subtropical areas in Africa, Asia & South America. It is found in Egypt in Kalyobia, Dakahlia, Sharkia, Cairo, Giza and Assiut.
Definitive Host & Habitat	Man, in tissues (extra-intestinal).	Man only, in lymph vessels and glands.
Reservoir host	Dog, horse, cattle	----------------
Diagnostic Stage	Larval stage: Size: 600×20 um Shape: comma shaped with rounded anterior end, long tapering tail and a rhabditiform esophagus.	Mainly microfilariae (but adults may be seen too): ● Sheath: loose ● Curves: smooth ● Periodicity: nocturnal in blood ● Size: 250 × 8 um ● Tail nuclei: free
Intermediate host	Cyclops	Mosquito (Culex, Aedes, Anopheles).
Infective Stage & Mode Of Infection	Infective larvae, through drinking water containing the infected cyclop.	Infective filiform larvae, when mosquito bites the man.
Life Cycle	Adults→ copulation→ male die→ female → migrate to subcutaneous tissue especially that become contacted with water→ during contact with water → uterus prolapses→ discharges larvae until they finished→ ingestion by cyclops→ body cavity→ moult twice→ infective larvae→ ingestion of cyclop by D.H. or R.H.→ larvae migrate through the wall of S.I.→ retro-peritoneal tissues→ maturation	Adults → in lymph vessels & glands of man (D.H.)→ microfilariae → blood → appear in peripheral blood by night (nocturnal periodicity) → mosquito during biting and sucking (I.H.) → cyclo-developmental transmission (just developing) → infective filiform larvae → go to man again during biting → it can enter by penetration or through bite wound or any abrasion → pass to lymph nodes and vessels → maturation → adults
Pathogenesis & Clinical Picture	1. Migration of female under the skin causes allergic reaction due to release of metabolic products→ urticarial rash, nausea, vomiting, diarrhea or asthmatic attack. 2. The skin opposite the anterior end shows red papule then blister which ulcerates. The worm lies in a subcutaneous tunnel & its course may be marked with induration and oedema. 3. 2ry infection of the ulcer leads to abscess, cellulitis and even septicemia. 4. Severe allergic reactions occur if the worm is broken during forced extraction & the larvae escape into the subcutaneous tissue.	1- Many infections are a symptomatic, & occur only in blood examination. 2- Main pathological features caused mainly by adult worms. 3- The disease pass in 2 phases: a. ***Acute inflammatory phase***: due to immunological reaction to toxic products of worms. 2ry infection by streptococci may be added. o Symptoms appear about one year after the infective bite. o Recurrent attacks of lymphangitis: affected vessels appear as raised, red hot, swollen & tender. Commonly in limbs especially in legs & genitalia (epididymo-orchitis & funiculitis). o Attacks of lymphadenitis: enlarged & tender regional lymph nodes. 2ry infection→abscess o Filarial or elephantoid fever: sudden onset with rigors & sweating lasts for few hours to several days & often recurs. o Bacterial & fungal super-infection. b. ***Chronic phase:*** o Hydrocele (most common): results from accumulation of straw colored fluid in sacs around testicles. o Obstruction of lymphatics: occurs slowly & usually follows years of repeated attacks of lymphangitis & fibrosis of lymph nodes & vessels by coiled worms inside lymphatics. o Distension & varicosities of lymphatics distal to obstruction o Persistent lymphatic edema. o Rupture of distended lymphatics: in pleural sac (chylo-thorax), peritoneal cavity (chylous ascitis), tunica vaginalis of testis (chylo-cele), intestine (chylous diarrhea) or in urinary tract (chyluria) with passage of microfilaria in urine. o Elephantiasis: ↑ permeability of the walls of obstructed lymphatics → leakage of lymph with high concentration of protein under the skin → proliferation of connective tissue & deposition of fibrous tissue. The skin & underlying tissues becomes hard, dense & non-pitting (hard edema). The skin appears thickened, rough & fissured susceptible to 2ry infection. There may be huge enlargement of the affected parts usually dependent one e.g. legs, scrotum, vulva, breast, & arms. Elephantiasis occurs after persistent high infection for 5 – 10 years.
Diagnosis	Clinically: ➢ The outline of the worm under the skin may be seen. ➢ Skin lesions: papule, blister & ulcer. Laboratory: ➢ Larvae are obtained by placing the affected part in cold water for few minutes. ➢ X-ray shows calcified females. ➢ Intradermal test & C.F.T. (Complement Fixation Test). ➢ Eosinophilia.	1- ***Clinical signs and symptoms***: above. 2- ***Laboratory investigations***: a. Recovery of microfilariae in blood at night. They are highest in capillary blood (ear lobe & fingers) o Examination of a drop of fresh blood shows movement of microfilariae. o Di-ethyl-carbamazine (DEC) provocative test: giving 100 ml & taking blood 45 minutes later. Thus, microfilariae can be demonstrated at any time of the day. o Concentration of microfilariae if they are scanty by Knott's technique: the sediment is examined for microfilariae. b. Detection of microfilariae in chylous urine from hydrocele: ether dissolves chyle. c. Detection of adults in lymph node biopsy. d. Immuno-diagnosis (serology): o Detection of circulating antigen: is of great value. o Detection of antibodies is of lower value because of cross reactivity (+ve in endemic areas). e. Molecular techniques: PCR f. High eosinophilia. 3- ***Imaging techniques***: a. Ultrasonography to visualize adults in lymphatics. Viable adults may be seen moving actively. b. Lymphoscintography will reveal lymphatic abnormalities especially dilatation of vessels.
Treatment	1. ***Removal of the worm***: o The ancient method: rolling the worm on a stick & pulling gradually each day until resistance is felt to avoid rupture of the worm. o Surgical removal. 2. ***Drugs***: anti-inflammatory drugs that help in expelling worms spontaneously or manually. o Thiabendazole (Mintezol) **OR** o Diethylcarbamazine (DEC, Hetrazan) **OR** o Metronidazole (Flagyl). 3. ***Symptomatic treatment***: antiseptic dressing, antibiotics, antihistaminics and corticosteroids.	1- Di-ethyl-carbamazine (DEC): the drug of choice. It kills adults and modifies microfilariae in a way that they are effectively removed by the host. The dose is given orally and repeated once every 6 months as long as the person remains microfilaraemic or has symptoms. It does not reverse the pathology already established but limits its progression. Antihistaminics & cortico-steroids are given to alleviate allergic reactions induced by the rapid destruction of the parasite 2- Ivermectin: effectively removes microfilariae from the blood, but does not affect adults. Thus, microfilariae reappear in the circulation. Treatment should be repeated half yearly or yearly. 3- Combine of DEC & Ivermectin: gives better results. 4- Symptomatic treatment: foot care, antibiotic and antifungal therapy to prevent and cure adenolymphangitis. Physiotherapy and banding to reduce and alleviate lymphoedema. 5- Surgical management: chronic hydrocele and elephantoid skin may be corrected surgically and should be preceded by a course of DEC.
Prevention & Control	1. Eradication of cyclops in wells by regular steaming or by chemicals as chlorine, copper sulphate & calcium oxide or breeding of fish that feed on them. 2. Boiling or filtering of well's water 3. Use of pumps.	1- Control of mosquito vector. 2- Mass treatment of patients to destroy microfilariae. ***N.B. See occult filariasis in page 13***

Name Parasite (Disease)	Brugia malayi (Malayan filariasis)	Loa loa : Eye African worm (Loaiasis or Loiasis)	Onchocerca volvulus (Onchocercosis or Onchocerciasis)	Mansonella perstans	Mansonella ozzardi
Geographical Distribution	Far east.	West and central part of tropical Africa.	Central Africa, Central & South America, Yemen & Saudi Arabia.	Central Africa, South America.	South America.
Definitive Host & Habitat	Lymph nodes & vessels of man.	Subcutaneous tissue of man.	Subcutaneous tissue on bony parts (in the form of nodules) of man.	Serous cavities & retroperitoneal tissue.	
Reservoir host	Monkeys and cats (for certain strains)	-----------	-----------	-----------	-----------
Microfilaria: Sheath	Loose	Tight	Unsheathed	Unsheathed	Unsheathed
Microfilaria: Size	250 × 8 um	250 × 8 um	300 × 10 um	100 × 5 um	200 × 5 um
Microfilaria: Curves	Kinky	Kinky	Smooth	Smooth	Smooth
Microfilaria: Tail nuclei	2 nuclei	Full	Free	Full	Free
Microfilaria: Periodicity	Nocturnal in blood	Diurnal in blood	Non-periodic in skin & subcutaneous tissue	Non-periodic in blood	
Vector (I.H.)	Mosquito (Mansonia mainly, Aedes & Anopheles)	Chrysops fly.	Simulium (also called black or buffalo fly). It is a daytime biting fly.	Cullicoides	
Infective Stage & Mode Of Infection	Infective filiform larvae, when infected mosquito bites man.	Microfilariae when the fly bites the man.	Microfilariae stage, when the man is bitten by the Simulium.	-----------	-----------
Life Cycle	As Bancroftian filariasis	<u>Adults</u> → subcutaneous tissue of man → <u>microfilariae</u> → appear in blood in day time (diurnal periodicity) → chrysops fly during biting → cyclodevelopmental transmission → man through biting again → subcutaneous tissue → mature	<u>Adults</u> → subcutaneous nodules → <u>microfilariae</u> → shedding → Simulium fly during biting → cyclodevelopmental transmission → man through biting again → subcutaneous tissue → maturation → <u>Adult</u>	-----------	-----------
Pathogenesis & Clinical Picture	**1-** Milder disease than Bancroftian filariasis. **2-** Elephantiasis affects legs below knees and arms below elbows. **3-** Genital involvement is rare. **4-** Chyluria is rare. **5-** Allergic manifestations are common.	**1-** **<u>Calabar swelling</u>**: most commonly observed on hands, wrists and forearms, but may appear anywhere in the body. They are painless and non-pitting. They last from few hours to several days and may recur for years. They are due to host's immune response to the parasitic antigens. **2-** **<u>Generalized</u>** pruritus, fatigue and arthralgia are common. **3-** **<u>Adult worms</u>** may be seen under the conjunctiva or skin & disappear in about 15 minutes leaving no trace. **4-** **<u>Serious complications</u>**: occur when microfilariae invade CNS, kidneys, heart.	**1-** ***Onchocerca nodule***: usually found over bony prominence as scalp, elbow, knee, ribs, iliac crests & scapula. They are firm, painless, rounded or oval, movable & vary in size from few mm in diameter to several cm. They do not cause medical problems unless they press on a vital organ. **2-** ***Eye lesions (River or Sudan Blindness)***: o <u>Cause</u>: due to toxic or allergic reactions to living & dead microfilariae migrating from nodules especially in the scalp. o <u>Manifestations</u>: keratitis, iridocyclitis, retinitis & optic neuritis. Subsequent fibrosis leads to complete blindness. o <u>Early symptoms</u>: photophobia, lacrimation, blepharospasm & foreign body sensation. **3-** ***Skin lesions***: o Severe dermatitis & edema at first then granuloma & fibrosis with severe itching. o Later on, loss of elasticity, atrophy & wrinkling of skin giving <u>premature senility appearance</u>. o In the groin it leads to hernia and hanging groin, which is composed of pendulous folds of skin that may contain enlarged lymph nodes. o De-pigmentation producing <u>leopard skin</u> or hyper-pigmentation (in Yemen; Sowda) with popular itchy eruptions	Usually non-pathogenic	
Diagnosis	As Bancroftian filariasis	**<u>Clinical</u>**: Worms seen under conjunctiva & history of Calabar swellings. **<u>Laboratory</u>**: o Detection of microfilariae in blood in day time (10 am – 2 pm). o Serology. o PCR. o Eosinophilia.	***Clinical manifestations***: above. ***Laboratory investigations***: ➢ <u>Demonstration of microfilariae</u> in aspirate or bloodless skin snips. ➢ <u>Biopsy</u> of nodules reveals adults. ➢ <u>Serological tests</u>: to detect antibodies. ➢ <u>Molecular techniques</u>: PCR. ➢ <u>Mazzotti test</u>: oral dose of DEC provokes intense pruritis within few hours due to death of microfilariae. Local application of it on skin is safer (called patch teat). Corticosteroids are given in severe reactions.	Blood film at any time for microfilariae.	
Treatment	As Bancroftian filariasis	**1-** Chemotherapy as in Bancroftian filariasis. **2-** Surgical removal of adult if seen under the conjunctiva.	**1-** Surgical removal of the nodules. **2-** Ivermectin (mectizan). **3-** DEC (Hetrazan).	➢ Unnecessary in asymptomatic cases. ➢ Ivermectin.	
Prevention & Control	As Bancroftian filariasis	**1-** Treatment of patients. **2-** Control of chrysops is difficult because it breeds in swampy areas of forests.	**1-** Treatment of patients. **2-** Control of Simulium fly is difficult, larvae & pupae attach to submerging rocks in rivers.	-----------	-----------

Occult filariasis; Tropical Pulmonary Eosinophilia (TPE)	
Definition	It is a filarial infection where the microfilariae are not found in blood but found with adult forms in tissues.
Cause	Immunologic hyper-responsiveness to microfilariae in the lung.
Characters	In the circulation: **1.** Absence of microfilariae from the circulation. **2.** High eosinophilia (> 3000/ul). **3.** Elevated titre of anti-filarial antibodies and IgE level. In the tissue: **1.** Presence of microfilariae surrounded by aggregates of eosinophils in the lungs. In X-ray there is diffuse miliary lesions in the lungs. **2.** Extrapulmonary lesions may occur as splenomegaly, lymphadenopathy and hepatomegaly. Presence of microfilariae surrounded by aggregates of eosinophils are seen too.
Clinical Picture	Paroxysmal cough, breathlessness & wheezing that is worse at night. There is impairment of lung function that may become irreversible in the chronic stage.
Treatment	DEC: leads to rapid suppression of the symptoms and reduction of number of eosinophils.

Class: Protozoa

General characters:

1- The protozoon: consists of:
  a. Protoplasm: consists of Outer ectoplasm &Inner endoplasm:
  b. Nucleus
2- Nutrition: through the ectoplasm by:
  a. Pseudopodia (amoebae).
  b. Cilia and flagella (Ciliates & Flagellates; Mastigotes).
3- Locomotion: by pseudopodia (amoebae), cilia (Ciliates) and flagella (Flagellates).
4- Excretion: by diffusion through body surface or by contractile vacuoles.
5- Secretions: include digestive enzymes, toxins, antigenic substances & cyst walls to resist unfavorable conditions.
6- Reproduction:
  a. Sexual: by
    i. Syngamy: Union of male & female gametes to form the zygote.
    ii. Conjugation: Exchange of nuclear materials between 2 organisms.
  b. Asexual: by
    i. Binay fission: nuclear division followed by cytoplasmic division (mostly all protozoa).
    ii. Endodyogeny: 2 daughter cells enclose in the cell membrane of the mother cell.
    iii. Schizogony:the nucleus divides into several nuclei followed by division of cytoplasm forming several segments (daughter cells) and give rise to a schizont.
    iv. Budding: the parent cell does not divide, but puts out a small budlike process (daughter cell) with its proportionate amount of chromatin; the daughter cell then separates to begin independent existence.

Classification of Protozoa

This classification is for reading only.

Phylum	Subphylum	Class	Members
Sacro-mastigo-phora	Sarcodina	Lobosea (Amoeba)	• *Entamoeba histolytica* • Commensal amoebae • Potentially pathogenic amoebae • Neglaria fowleri • Acanthamoeba: o Granulomatous Amoebic Encephalitis o Acanthamoeba keratitis
	Mastigo-phora	Zoomastigo-phora (Flagellates)	• *Giardia lamblia* • *Trichomonas vaginalis* • *Leishmania* ○ Cutaneous ○ Visceral • *Trypanosoma*: ○ African ○ American
Ciliophora	---------	Kinetofragmino-phora (Ciliates)	• *Balantidium coli*
Apicomplexa	---------	Sporozoa	• *Cryptosporidium parvum* • *Cyclospora cayetanensis* • *Isospora belli* • *Plasmodium* • *Babesia* • *Toxoplasma*
Microspore	---------	Microspore	• Microsporae

Classification of Protozoa According to Habitat

This classification is for understanding & studying.

Luminal	Intestinal	Small intestine	➢ *Giardia lamblia* ➢ *Cryptosporidium* ➢ *Isospora* ➢ *Cyclospora* ➢ *Microsporidia* (affecting small intestine)
		Large intestine	➢ *Entamoeba histolytica* ➢ *Balantidium coli*
	Urogenital		➢ *Trichomonus vaginalis*
Blood protozoa	Haemo-flagellates		➢ *Visceral Leishmania (L. donovani complex)* ➢ African trypanosomes: o *T. gambiense* o *T. rhodeseinse* ➢ American trypanosomes: o *T. cruzi*
	Intra-erythrocytic sporozoa		➢ *Plasmodium:* o *P. vivax* o *P. ovale* o *P. malariae* o *P. falciparum* ➢ *Babesia*
Tissue protozoa			➢ Cutaneous *leishmania* o Old world (OWCL): ▪ *L. tropica* ▪ *L. major* ▪ *L. aethiopica* o New world (NWCL): ▪ *L. peruviana* ▪ *L. braziliensis* ▪ *L. Mexicana* ▪ *L. pifanoi* ➢ *Toxoplasma gondii* ➢ Free living amoeba (FLA): o *Naegleria fowleri* o *Acanthamoeba* species: ▪ Granulomatous Amoebic Encephalitis (GAE) ▪ Acanthamoeba keratitis ➢ *Microsporidia*

Parasite (Disease)	Entamoeba histolytica (Amoebiasis)	Balantidium coli (Balantidiasis)
Geographical Distribution	Cosmopolitan (more in areas of overcrowding and bad sanitations)	Cosmopolitan, widely distributed In pig raising areas.
Definitive Host & Habitat	Man in the wall and lumen of colon especially caecum and sigmoido-rectal region	Lumen, mucosa and sub-mucosa of large intestine of man, especially the caecum.
Reservoir Host	------------------------	Pigs
Diagnostic Stage	Cyst or Trophozoite stage	Cyst or Trophozoite stage
Infective Stage & Mode Of Infection	Quadrinucleate (mature) cyst, through: • Auto-infection • Hetero-infection: o Eating raw vegetables fertilized with human faeces. o Open source of water contaminated with human excreta. o Flies & cockroaches carrying cysts to food or drink. o Food handlers, especially chronic asymptomatic cyst carriers.	Cyst stage, through: • Auto-infection. • Ingestion of cysts with food or water contaminated by pig's or human excreta.
Life Cycle	Quadrinucleate cyst → ingestion → pass stomach acidity → small intestine → lower ileum → excystation → 8 small amoebae → multiply by binary fission →EITHER: remain in the lumen → feed on starch & mucus OR invade the wall by their lytic enzymes → flask-shaped ulcers. (In chronic cases, they produce cysts and pass with faeces).	Cyst → ingestion → small intestine → excystation → single parasite → multiplication → EITHER remain in the lumen with no symptoms OR invade the intestinal wall → produce flask-shaped ulcers → after period → encystment → cysts in stool.
Pathogenesis	There are 2 forms of E. histolytica. They are similar in morphology & can inter-change in certain condition.: **1-**Pathogenic or tissue form. **2-**Non-pathogenic or lumen form. The pathogenic activity of E. histolytica depends on: **1-**Virulence of organisms. **2-**Resistance of the host. **3-**Condition of the intestinal tract. The amoebae secretes histolytic enzymes produce necrosis of the intestinal mucosa with rapid lateral & downward extension of ulceration → ***flask-shaped ulcer*** (wide base & narrow opening). This is followed by: **1.**Proliferation of connective tissue, which lead to fibrous thickening of intestinal wall **2.**Intensive ulceration may accompanied by 2ry bacterial infection. **3.**Extra-intestinal invasion: mainly to liver.	**1-**Invasion of the mucosa is affected by: **a.** Cytolytic enzyme hyaluronidase. **b.** Boring action of cilia. **2-**Secondary bacterial infection may follow the invasion. This leads to formation of ***flask-shaped ulcers*** as in acute amoebic infection with signs and symptoms of dysentery. **3-**Extra-intestinal spread is rare. **4-**Complications: **a.** Hemorrhage. **b.** Perforation. **c.** Peritonitis. **d.** Appendicitis.
Clinical Picture	***Asymptomatic Infections:*** The most common type. The parasite is found in the lumen with no invasion of mucosa and pass with stool (cyst passer). ***Symptomatic Infections:*** **Intestinal Amoebiasis:** Acute stage: • The onset is gradual with dysentery, abdominal pain & tenderness. • Tenesmus, painful spasms of anal sphincter is a sign of ulcerations. • Stool contains blood, mucus, shreds of necrotic tissue & trophozoites. Chronic stage: • Recurrent attacks of dysentery with intervening GIT disturbances & constipation. • Localized tenderness. • In long standing infections, there is loss of weight & cachexia. **Extra-intestinal Amoebiasis:** Hepatic Amoebiasis: • Amoebic hepatitis: enlarged tender liver with pain in the right hypochondrium. • amoebic liver abscess: enlarged tender liver and leukocytosis. Taping of abscess reveals thick, ***anchovy-sauce or chocolate-colored pus*** containing trophozoites. Pulmonary Amoebiasis: • Fever, leukocytosis & evidence of consolidation. • Trophozoites may appear in the sputum in case of bronchial erosion. **Complications:** **1.**Amoeboma: granuloma around the ulcer. It may be confused with malignant tumors. **2.**Haemorrhage: due to erosion of a blood vessel in the intestinal wall. **3.**Perforation of an amoebic ulcer→ peritoneal infection (peritonitis). **4.**Stricture: due to healing by fibrosis. **5.**Appendicitis.	
Diagnosis	***Intestinal Amoebiasis:*** **Clinical:**See above (N.B. it should be differentiated from bacillary dysentery). **Laboratory:** *Direct stool examination*: Bulky, offensive, acidic, scanty exudate: reveals blood, epithelial cells, Charcot Leyden crystals & amoebae are present. ▪ Examination for the trophozoites: - Wet preparation using saline, reveals highly refractile shining bodies with progressive directional crawl and ingested red blood cells. - Stained smears will reveal the morphology - Permanent preparation using iron heamatoxylin or trichrome stain. ▪ Examination for the cysts: - Smear stained with iodine or trichrome. - Concentration techniques. *Sigmoidoscopy (invasive):* To visualize lesions or take a biopsy. *Indirect diagnosis:* ▪ Radiological (barium enema). ▪ Serological. ▪ Detection of copro-antigen in stool. ▪ Molecular techniques. ***Extra-intestinal Amoebiasis:*** **Clinical:** According to the organ affected. **Laboratory:** • Aspirate examination from lung or liver abscess for trophozoites if accessible. • Liver scanning. • Radiology for diaphragm level and pulmonary lesions. • Serology. • Leukocytosis can be found due to 2ry bacterial infection.	**Clinically:** • Clinical manifestations of dysentery. **Laboratory:** • Finding trophozoites in diarrhoeic stool and cysts in formed stool. • Stool should be examined several times since discharge of the parasite is intermittent.
Treatment	Intestinal Amoebiasis: • Metronidazole (Flagyl) followed by Diloxanide fluroate for 10 days each. **OR** • Tinidazole (Fasigyn) for 2 – 3 days followed by Diloxanide fluroate for 10 days. Extraintestinal Amoebiasis: • Metronidazole **OR** Tinidazole followed by Diloxanide fluroate. • Aspiration of abscesses or open surgical drainage may be needed.	• Metronidazole (Flagyl) **OR** • Oxytetracycline
Prevention & Control	**1-** Treatment of patients. **2-** Food handlers should be examined and treated. **3-** Environmental sanitation. **4-**Human faeces should not be used as fertilizers. **5-**Personal prophylaxis.	The same as in amoebic infections, plus: Care of pig's excreta is of great importance where they are raised.

Parasite (Disease)	Giardia lamblia (Giardiasis)	Cryptosporidium parvum (Cryptosporidiasis)	Cyclospora cayetanensis (Cyclosporiasis)	Isospora belli (Isosporiasis)	Trichomonas vaginalis (Trichomoniasis)
Geographical Distribution	Cosmopolitan, more in warm climates	Worldwide	Worldwide	Worldwide	Cosmopolitan
Definitive Host & Habitat	The duodenal mucosa, upper part of small intestine, bile duct & gall bladder of man, especially children	Small intestine of man, intracellular within the brush border	Enterocyte (intracellular) of upper part of small intestine of man	Intracellular of epithelial cells of small intestine of man	Man (vagina , urethra , prostate)
Diagnostic Stage	Cyst or Trophozoite stage	Oocyst stage	Unsporulated oocyst stage	Oocyst stage	Trophozoite only
Infective Stage & Mode Of Infection	Cyst stage, through: • Auto-infection • Hetero-infection: ▪ Contaminated food (vegetables) by Musca fly or food handlers. ▪ Contaminated water.	Sporulated oocyst, through: • Auto-infection: ○ Thin-walled →endogenous ○ Thick-walled →exogenous • Ingestion of contaminated food or drink	Mature sporulated oocyst, through ingestion of: • Contaminated vegetables & fruit. • Contaminated chlorinated & filtered water arising from water-storage tanks.	Sporulated oocyst, through: • Auto-infection. • Ingestion of contaminated food or drink.	Trophozoite stage, through sexual intercourse, directly from an infected partner.
Life Cycle	Cyst→ duodenal lumen → excystation →multiply by longitudinal binary fission → attach to the mucosa → some encyst & pass in stool	**Merogony**: asexual reproduction → meronts (contains merozoites) → initiate new cycles adjacent cells **Gametogony**: sexual reproduction → gamonts → contains micro &macrogametes → fertilization → zygotes → thin- & thick-walledoocysts → infection (thin walled can cause endogenous infection)	• Not completely known • Asexual & sexual cycles are believed to occur • Unsporulated, immature, non-infective oocysts are shed in patient's faeces. • Sporulation occurs outside the host (5 days) →mature, sporulated & infective oocyst (No autoinfection).	Oocyst → small intestine → sporozoites →penetrate epithelial cells → immature sporulated oocyst → sporulation (may occur in the lumen or outside the host)	------------------
Pathogenesis and Clinical Picture	1-Asymptomatic if the parasite just feed on mucus. 2-Symptomatic cases: Hyperaemia & duodenatis, manifested by ▪ Epigastric pain ▪ Disturbances of digestion ▪ Diarrhea & flatulence 3-Severe symptoms: as: ▪ Persistent diarrhea, steatorrhea, hypoproteinaemia & fat-soluble vitamin deficiencies. ▪ Mal-absorption due to villus atrophy. ▪ Cholangitis & cholecystitis may lead to jaundice & colic pain ▪ Occur in patients with impaired immunity such as: ▪ Hypo-γ-globulinaemia ▪ Diminished secretory IgA in SI ▪ Dimished gastric acidity or achlorohydria	1- Inflammatory changes: villous atrophy and crypt hyperplasia. 2- In immuno-competent subject: ▪ Mild self-limited diarrhea lasting for 2 weeks. ▪ In some cases, especially in children, the condition is accompanied by abdominal discomfort, anorexia, fever, nausea & loss of weight. 3- In immuno-compromised subject: ▪ The disease is severe, especially in AIDS patients & could be life-threatening (opportunistic infection). ▪ Severe diarrhea, *malabsorption* → dehydration. ▪ Dissemination of the parasite to other organs can develop, as esophagus, gall bladder, urinary bladder, & respiratory tract.	1- Inflammatory changes: villous atrophy and crypt hyperplasia of affected area. 2- In immuno-competent subject: • Watery-diarrhea that tends to occur in a *relapsing or cyclical* pattern. Accompanied by nausea, vomiting, flatulence & abdominal cramps. • May cause anorexia, loss of weight, fatigue & low-grade fever. 3- In immuno-compromised subject: In AIDS patients, the illness is severe, prolonged & tend to recur. Biliary affection may develop.	**1.** Inflammatory changes: villous atrophy and crypt hyperplasia of affected area. **2.** In immuno-competent subject: ▪ Often asymptomatic. ▪ Self-limited diarrhea may develop ▪ Chronic diarrhea with abdominal cramps are seen in some cases **3.** In immuno-compromised subject (opportunistic infection): Severe diarrhea with malabsorption and dehydration can develop and be life threatening.	**In women:** ▪ Trophozoites found in vagina & urethra → feed on the mucosal surface→ sloughing of sq. epith. cells ▪ 50% are asymptomatic. The rest suffer from profuse odorous discharge associated with burning, itching, dyspareunia, frequency of urination & dysuria ▪ On examination: diffuse vulval erythema , excessive discharge & vaginal wall inflammation **In Men:** Frequently asymptomatic When infection involves prostate or higher urogenital tract, symptoms may appear as: thin discharge, dysuria & nocturia ± enlarged prostate & epididymitis.
Diagnosis	**Clinically**: suggestively **Laboratory**: ➢ Direct stool analysis: reveals • Trophozoite in diarrheic stool • Cyst &trophozoite in formed stool. • Concentration technique gives higher positivity. • If the result is –ve the test should be repeated again after some days because the excretion of the parasite is irregular. ➢ String test (Enterotest) ➢ Indirect through: • Serological tests (of little value) • Detection of copro-antigen.	**Clinically**: as above. **Laboratory**: ➢ Stool analysis: Simple smear & concentration methods (Sheathter's sugar floatation) are used. Oocyst can be detected by: ▪ Staining by acid-fast stain (MZN stain). ▪ Immunofluorescence assay. ➢ Intestinal biopsy: To detect meronts and gamonts.	**Clinically**: as above. **Laboratory**: ➢ Stool analysis: the specimen can be examined fresh unstained or stained with acid-fast stain (MZN stain). ➢ Jejunal biopsy: to detect asexual stages.	**Clinically**: as above. **Laboratory**: Stool analysis: The oocyst can be seen in fresh unstained or stained with acid-fast stain (MZN stain).	**1.** Microscopic examination of wet films prepared from discharge or urine. **2.** Culture of discharge on suitable media (as Modified Diamond's medium) when microscopy fails. **3.** Detection of antigens by immunological tests as: ▪ Enzyme immunoassay ▪ Direct fluorescent antibody test using labeled monoclonal antibodies **4.** Molecular techniques as DNA probe.
Treatment	• Metronidazole (Flagyl) **OR** • Tinidazole (Fasigyn) • Albendazole, recently • Nitazoxanide	• In immuno-competent patients: self-limited • In immuno-compromised patient: ▪ Nitazoxanide ▪ Fluid & electrolyte replacement	Trimethoprim **combined with** Sulphamethoxazole	• Trimethoprim **combined with** Sulphamethoxazole • Fluid & electrolyte replacement	• Metronidazole (Flagyl • Treatment of the sexual partner
Prevention & Control	As Amoebiasis	**1.** Measures against faeco-oral transmission **2.** Proper filtration of drinking water **3.** Boiling of drinking water to immuno-compromised patients.	Infection can be prevented by proper washing of vegetables and fruits and boiling of water.	Measures against faeco-oral transmission	**1.** Treatment of patients & their partners. **2.** Diagnose &ttt of asymptomatic carriers. **3.** Use of condoms is very effective.

Parasite (Disease)	Visceral Leishmania (Kala-azar, Dum-dum fever, black fever)		Trypanosoma		
			African (Sleeping Sickness)		American (Chaga's disease)
	Old world Leishmaniasis: L. donovani & L. infantum	New world Leishmaniasis: L. chagasi & L. amazonensis	T. brucei gambiense	T. brucei rhodesiense	T. cruzi
Geographical Distribution	L. donovani: Southeast Asia & central Africa. L. infantum: Mediterranean area, Europe & Africa.	Central & south America.	West Africa & western part of tropical Africa	East & central part of tropical Africa	Central and south America
DH & Habitat	Man in Reticulo-Endothelial System		Man, less in blood	abundant in blood	Man, domestic & wild animals
Reservoir Host	--------------------		Goats, cattle & pigs	Wild game animals	-----------
Diagnostic Stage	Amastigote in biopsy and promastigote in culture.		Multi form trypanosomes		C-shaped trypanosomes
Vector	Sand fly: Phlebotomus	Sand fly: Lutzomyia	Tsetse fly: Glossina palpalis	Jungle Tsetse fly as: Glossina morsitans	Winged bug: Triatoma & Rhodnius
Infective Stage & Mode Of Infection	Promastigote stage, through bite of sand fly & inoculation of promastigotes.		Short stumpy metacyclic trypanosomes, through bite of tsetse fly		As African but through contamination of the site of bite or mucous membranes with faeces
Development inside Vector (Mechanism of Disease Transmission)	Amastigote → taken by female sand fly by bite→ change into promastigote → multiply by longitudinal binary fission → migrate back to the buccal cavity → infect another host during biting (Biological transmission)		Multi form trypanosomes→ taken by tsetse fly by bite→ change into amastigote → midgut → longitudinal binary fission → migrate back to the salivary gland→ multiply → epimastigote→ short stumpy metacyclic trypanosome→ infect another host during biting (Biological transmission)		As African but: ▪ Pass to the hindgut. ▪ Don't go to the salivary gland ▪ Infective stage pass with faeces
Pathogenesis and Clinical Picture	1. Promastigotes that engulfed by skin macrophage transforms to amastigotes and start multiplication. 2. A local papule (leishmanioma) are rarely seen but described in children. 3. The parasites are present in few numbers in blood & are taken by reticulo-endothelial system cells & other organs that show hyperplasia. 4. The onset is usually gradual with initial fever (intermittent with double daily rise). 5. Diarrhea & dysentery are common. 6. Splenomegaly, hepatomegaly & lymphadenopathy are seen. 7. Invasion of bone marrow results in aplastic anemia, leukopenia & thrombocytopenia. There is reversal of albumin/globulin ratio due to elevation of gamma-globulins. 8. Skin changes may occur in the form of: dark pigmented erythematous areas or depigmented macules distributed over the body. A butterfly distribution over the nose is common. 9. Post-kala-azar dermal leishmaniod in the form of depigmented skin nodules may develop due to spontaneous arrest of the disease or incomplete treatment with antimony compounds. 10. Weight loss and emaciation (abnormally thin & weak), which render the patient to secondary infections. This may lead to death.		***Chronic course*** (≥ 3 years) with these stages: 1. Incubation period (≥ 14 days). 2. Trypanosomal chancer: local inflammatory nodule at the site of bite (lasts 1-2 weeks). 3. Parasitaemia: trypanosomes pass to the blood & multiply → irregular fever, headache, joint & muscle pain and rash. 4. Invasion of lymph nodes: especially posterior cervical region (***Winterbottom's sign***) → generalized weakness, hepatomegaly, splenomegaly, irregular erythematous rash & anaemia. The trypanosomes are present in lymph nodes, blood & bone marrow. 5. Invasion of the CNS: ***gradually*** with perivascular cellular infiltration → ischemic softening of tissues & petechial haemorrhages→ diffuse meningioencephalitis & meningiomyelitis. There is mental apathy & retardation, slow speech, tremors, involuntary movements & convulsion. Lastly, the sleeping stage develops & the patient falls into coma. Death occurs either from the disease or from inter-current infection.	▪ ***More rapid & fatal course*** even before the appearance of neurological manifestations. ▪ *The incubation period is short*. ▪ Bouts of fever & rigors are more frequent & severe. ▪ It appears in blood early in infection & in abundant numbers. ▪ Myocarditis & emaciation are prominent. ▪ ***CNS is involved early***. ▪ Untreated patient die within one year after infection.	**The acute form:** 1. Organisms proliferate at the site of infection and produce erythematous indurated area called ***Chagoma***, which occur frequently in the face. 2. Organisms spread rapidly to the regional lymph nodes → blood → organs and tissues. They usually appear in phagocytes of liver (Kuppfer cells), spleen & cardiac muscle. 3. Sudden unilateral edema of the eye lids without conjunctivitis (***Romana's sign***). 4. Signs and symptoms of generalized infection: high fever, muscle pain & exhaustion. Epistaxis is more common in young children. 5. Generalized glandular edema, hepatosplenomegaly and rarely skin rash. 6. In severe infections, there are signs of meningioencephalitis & cardiac involvement and heart failure. 7. Complications include: death, chronicity or recovery. **The chronic form:** More common in adults & manifestations depend on the site invaded. 1. In the heart: ECG changes with signs & symptoms of heart failure. 2. Dilatation of parts of the GIT: as mega-oesophagus & mega-colon manifested by dysphagia & constipation. 3. Invasion of CNS or thyroid gland is commonly less. 4. Immunosuppression results in exacerbation of the infection.
Diagnosis	1. **Clinically**: as above especially fever with double daily rise, leukopenia & splenomegaly. 2. **Detection of the parasite in the blood or material from the spleen, liver, bone marrow or lymph nodes**: specimens are examined by 3 methods: a. Microscopy: blood examined by thick drop preparation or by buffy coat method. Smears are stained with Giemsa stain. b. Culture of specimens on suitable medium. c. Intra-peritoneal inoculation of material in hamsters. 3. **Immunodiagnosis**: a. ***Montenegro (leishmanin) test***: gives delayed reaction (after 3 days). Usually negative in active infection & becomes positive after successful treatment. b. Serological tests.		• **Clinically**: as above. • **Demonstration of trypanosomes** in aspirate from chancre, lymph nodes, blood, CSF by : ○ Microscopic examination of fresh unstained or stained films ○ Culture on suitable medium. ○ Animal inoculation fails unless the animal is irradiated to decrease its immunity. • **Detecting an increase in total serum IgM level**: always elevated due to antigenic variation of the surface coat of the organisms. • Serological tests: not available commercially.	As T.brucei gambiense but: ▪ More frequently appear in blood. ▪ More readily demonstrated with animal inoculation with appearance of posterior nuclear shift.	1. Demonstration of the parasite in the blood by: a. Direct thick smear b. Special concentration techniques. c. Culture on suitable medium. d. ***Xenodiagnosis***: feeding laboratory-bred winged bug on the patient's blood then examining the gut of the bug for trypanosomes. 2. Serodiagnosis 3. Biopsy of enlarged lymph nodes may reveal amastigotes. 4. ***Cruzin test***: intradermal test, gives delayed reaction +ve cases. 5. Molecular techniques as PCR
Treatment	1. Antimony sodium gluconate (Pentostam) 2. Pentamidine 3. Allopurinol (for AIDS patients)		1. In the early stages: ● Pentamidine ● Suramin 2. In the late stages: ●Trypasamide 3. For both stages: ● Eflornithine (Ornidyl)		1. Nifurtimox: it inhibits intra-cellular growth 2. Primaquine: destroy trypanosomes in blood
Prevention & Control	1. Treatment of the patients. 2. Control of vector. 3. Protection by using wire screens, mosquito or repellents. 4. Vaccination in endemic areas using suspension of living promastigotes resulting in lasting immunity.		1. Treatment of patients. 2. Control of vectors. 3. Chemoprophylaxis: one dose of Pntamidine every 5 – 6 months.		1. Detection and treatment of cases. 2. Control of vector.

Parasite (Disease)	Plasmodium (Malaria)				Babesia Species (Babesiosis)
	P. vivax (Vivax or Benign tertian malaria)	**P. ovale (Ovale or Oval tertian malaria)**	**P. malariae (Malariae or Quartan malaria)**	**P. falciparum (Falciparum, Subtertian or Malignant malaria)**	
Geographical Distribution	World-wide	Tropical area	Subtropics & temperate zones	Tropical area	Europe, North & South America
Diagnostic Stage	Ring, trophozoite, schizont, gametocyte stages in infected RBCs, but with P. falciparum ring & gametocyte stages only.				Merozoites
Vector	Female Anopheles Mosquito				Hard ticks
Infective Stage & Mode Of Infection	Sporozoite stage, through bite of female Anopheles mosquito & inoculation of sporozoites. Merozoite stage, through blood transfusion.				Sporozoites, through bite of hard tick inoculating them
Life Cycle	**In man:** **N.B.** Some P. ovale & vivax enter asexual multiplication exo-erythrocytic. Infected Mosquito → Sporozoites → 40 minutes in blood → Hepatocyte → Hypnozoites (resting stage)→ Trophozoites → Schizonts → rupture → Liver Merozoites → Blood → RBCs → Ring stage → Ring stage → Tropozoites → Schizonts (reythrocytic) → rupture → 6 – 36 Blood Merozoites → Some Merozoites form Gametocytes → Mosquito during biting (man become infective to mosquito) **In Mosquito Vector:** Gametocytes (♂→exflagellation→ microgamete & ♀→macrogamate→ fusion (**sexual**)) → Zygote → Ookinete → Oocyst → Sporocyst → Salivary glands → Sporozoites → man during biting (mosquito become infective to man)				Sexual multiplication in hard tick → sporozoites → salivary glands → bite → RBCs (directly) → asexual multiplication by budding → merozoites → release → other cells
Pathogenesis and Clinical Picture	**1.** Infection starts with incubation period (liver phase) followed by prodromal symptoms which are influenza like. **2.** Then malarial paroxysms, which coincides with: a. Rupture of RBCs (due to ↑ osmotic fragility). b. Liberation of metabolites of the parasites. c. Immunologic response to the parasitic antigens. **3.** Malarial paroxysms (clinical attacks), include 3 stages: a. ***Cold stage***: the patient complains of sudden chill, extreme cold & his temperature is elevated (lasts 10 – 15 minutes). b. ***Hot stage***: headache, high fever & hot dry flushed skin (lasts 2 – 6 hours). c. ***Sweating stage***: profuse sweating, temperature falls & the patient is weak & exhausted (may last for several hours). **4.** Malarial paroxysms is repeated as follows: a. Every 48 h in P. vivax & P. ovale → tertian malaria. b. Every 72 h in P. malariae → quartan malaria. c. Every 36-48 h or irregular in P. falciparum → subtertian malaria. ▪ It is repeated for 2 weeks with ↓ intensity thin stop ▪ Its termination may mean elimination of infection but relapse or recrudescence may presented : ***Relapse***: recurrence of attacks due to reactivation of hypnozoites in liver (occurs with P. ovale & vivax) ***Recrudescence***: recurrence of attacks in patients having low-grade parasitaemia when they become debilitated (occurs with P. malariae & falciparum). ▪ No relapse with blood transfusion. **5.** Anemia: due to destruction of RBCs. Merozoites of P. vivax & P. ovale invade reticulocytes only. Merozoites of P. malariae invade ***old RBCs***. This restricts the infection. However, those of P. falciparum invade ***RBCs of any age*** causing severe hemolytic anemia. **6.** Hepatosplenomegaly: because of enhanced phagocytosis of RBCs remnants & other debris. ***Complications:*** **1.** P. vivax, ovale & malariae malaria are relatively benign. **2.** Chronic P. malariae infection results in immuno-complex deposition on the glomerular walls → nephrotic syndrome. **3.** P. falciparum is usually severe & fatal (thus called Pernicious): **a.** In this infection, the parasitized RBCs develop knobs on their surface. So they adhere together & to specific receptors on the endothelial cells of the capillaries of internal organs → partial occlusion → anoxia & necrosis which may be fatal. The clinical picture differs according to the site of occlusion: ▪ Cerebral malaria: severe headache, drowsiness, confusion & coma ▪ Dysenteric malaria: abdominal pain, vomiting & bleeding ▪ Pulmonary edema ▪ Algid malaria: Because of pulmonary edema, GIT bleeding or gram –ve septicemia. Hypotension rapidly develops → impaired capillary perfusion, vascular collapse & shock. ▪ Hypoglycemia: may result from impaired hepatic gluconeogenesis. ▪ Renal failure: Renal anoxia → acute renal failure. **b.** Hyperactive malarial splenomegaly: characterized by chronic splenomegaly with marked elevation of IgM. This may be due to reduction of suppressor T-cells that control B-cell activation in P. falciparum infection. **c.** Black water fever: may be the result of repeated attacks of P. falciparum infection & incomplete quinine therapy. Massive intravascular haemolysis occurs with anemia, jaundice & hemoglobinuria (dark red or black urine). The cause may be autoimmune with development of antibodies to the infected RBCs.				**1.**Invasion of RBCs → their rupture. **2.**Infection is asymptomatic or mild and self-limited in most cases. **3.**It is characterized by fever (not periodic), chills, headache, myalgia & backache. **4.**Mild to moderate haemolytic anaemia → jaundice. **5.**More severe forms with rapidly progressive illness characterized by fever, anaemia, jaundice & renal failure may develop. **6.**In splenectomized patient, the infection is fulminating & may be fatal.
Diagnosis	**1.** Examination of thin and thick blood smears from the patient during the febrile conditions. This shows ring, trophozoite, schizont and gametocyte stages of the parasite. In P. falciparum, only rings & gametocytes are seen. RBCs harbouring trophozoites & schizonts of P. falciparum are trapped in blood capillaries of the internal organs (due to the surface knobs they developed). **2.** Detection of the circulating parasite antigens using monoclonal antibodies. **3.** Use of DNA and RNA probes.				**1.**Blood film examination: reveals multiple small rings in RBCs that can be *differentiated from P. falciparum by absence of malaria pigment.* **2.**Serology: is useful especially in presence of low parasitaemia. **3.**PCR
Treatment	***Classification of drugs that treat human malaria***: **1.** Drugs that destroy parasite stages in the liver (tissue schizonticides): Primaquine (kills hypnozoites too). **2.** Drugs that destroy parasite stages in the blood (blood schizonticides): Quinine, Chloroquine & recently Artimesinin (plant extract). **3.** Drugs that destroy gametocytes in the blood (blood gametocyticides): a. Chloroquine kills gametocytes of P. vivax, P. ovale & P. malariae. b. Primaquine kills all gametocytes of all four species. Thus they render the patient non-infectious to the mosquito. ***The recommended regimens for malaria treatment***: **1.** Treatment of clinical attacks: Chloroquine. **2.** Radical treatment: Primaquine is given after treatment of clinical attack to kill hypnozoites of P. vivax & P. ovale. **3.** Anti-malarial chemoprophylaxis: for healthy persons entering a malaria endemic area: **a.** Causal prophylaxis as Primaquine. **b.** Suppressive prophylaxis as Chloroquine during stay in malaria endemic area. **4.** Drug resistant malaria: is overcome by drug combination as **Coartem** (artemether and lumifantrine) is recently developed.				**1.**Combination of Clindamycin & Quinin. **2.**Blood transfusion in severe cases.
Prevention & Control	**1.** Early detection and treatment of human cases. **2.** Control of mosquito vector **3.** Chemoprophylaxis of healthy human entering a malaria endemic area. **4.** Vaccination against malaria. A vaccine is already tested in South America and some parts of Africa with promising results. ***In Egypt***, malaria is under control. However it is threatened by falciparum malaria from neighboring countries especially Sudan where falciparum malaria is endemic.				Tick control measures prevent infection.

Parasite (Disease)	Cutaneous Leishmania (Cutaneous Leishmaniasis; CL)						
	A-Old World Cutaneous Leishmaniasis (OWCL)			B-New World Cutaneous Leishmaniasis (NWCL)			
	L. tropica (Dry or Urban CL)	**L. major (Wet or Rural CL)**	**L. aethopica**	**L. peruviana (Uta)**	**L. brazilinesis (Espundia)**	**L. maxicana (Chiclero ulcer, Bay sore)**	**L. pifanoi**
Geographical Distribution	Middle East, Asia & Africa in people living in big cities.	Middle East, Asia & Africa in rural areas.	Ethiopia & Kenya	Central & south America.			
Vector	Snad fly (Phlebotomus fly species)			Snad fly (Lutzomyia fly species)			
Diagnostic Stage	▪ Amastigote in the early lesion biopsy. ▪ Promastigote in culture.						
Infective Stage & Mode Of Infection	Promastigote form, through bite of(Phlebotomaus , lutzomyia) fly.						
Development Inside the Vector	<u>Amastigote</u>→ taken by female sand fly by bite→ change into <u>Promastigote</u> → multiply by longitudinal binary fission → migrate back to the buccal cavity → infect another host during biting (<u>Biological transmission</u>).						
Pathogenesis and Clinical Picture	**1.** The lesion develops in exposed parts of the body, usually face. It may be single or multiple. **2.** At the site of bite, there is a localized nodule due to multiplication of the organisms in the skin macrophages and granulomatous reactions around them. **3.** The lesion is dry and ulcerated only after several months giving an ulcer with cut edges, raised indurated margin and scanty exudate. It is known as ***Oriental sore***. **4.** 2ry bacterial infection is common, the ulcer <u>heals spontaneously</u> if untreated after about one year leaving a disfiguring scar.	1. It produces an <u>acute</u> infection with duration of 3-6 months. 2. The lesions occur 1ry on the lower limbs. 3. The lesions are moist with serous exudate and tend to ulcerate very early. • ***Resistance*** to re-infection with the same species (L. tropica or major) following a 1ry infection is strong and long lasting.	• It produce <u>diffuse</u> CL . • The parasite proliferate indefinitely forming many lesions. • This probably the result of not only deficient cell-mediated immunity but also of some characteristics of the parasite itself. • Lesions resemble lepromatous leprosy.	• Disease known locally as ***Uta.*** • May one or small number of skin lesions that are <u>self-healing</u> similar to L. tropica.	• Also, produce skin ulcers. • Lymphatic spread result in wide spread ulcerations & involvement of mucous membranes & can cause erosion of the nasal septum, palate or larynx with oedema, tissue destruction & 2ry bacterial infection. • This result in great deformity that is called ***Espundia***.	• Produce ***Chiclero ulcer or Bay sore.*** • Usually single & affects the <u>ear</u> causing destruction of cartilage. • Occurs in the forest workers who collect the chicle gum. • <u>Rarely</u> cause diffuse cutaneous lesions with nasopharyngeal mucosal involvement & regional lymhadenopathy.	• The initial lesion is single then spreads slowly like lepromatous leprosy. • <u>*Does not ulcerate or heal.*</u>
Diagnosis	**a.** <u>Clinically</u>. **b.** <u>Detection of the parasite</u> **:** ▪ Examination of material aspirated or scraped from the <u>edge</u> of lesion. A biopsy could be taken & submitted for histologic examination. <u>Amastigotes</u> can be seen & are numerous in the early than in late lesions . ▪ Culture on suitable medium may demonstrate <u>promastigote</u> forms. **c.** ***Montenergo test***: is an intradermal test using <u>antigen</u> from cultured promastigotes. It is +ve in more than 95% of infections. It gives a delayed reaction that appears as an indurated area after 3 days. **d.** <u>Serological tests</u>.						
Treatment	<u>***Systemic treatment***</u>: ▪ Pentavalent antimony as antimony sodium gluconate or pentostam. ▪ Pentamidine is given in diffuse cutaneous leishmaniasis caused by L. aethiopica. ▪ Allopurinol, Ketoconazole and Dapsone are proved useful.			<u>***Local treatment***</u>: ▪ <u>Physical methods</u> as surgical excision, curettage, heat and freezing therapy. ▪ <u>Chemical methods</u> using topical preparations as 2% chlorpromazine and clotrimazole 1%. ▪ <u>Intradermal injection</u> of interferon gamma around the lesion promotes healing of ulcers.			
Prevention & Control	**1-**Treatment of patients. **2-**Control of vector. **3-**Protection by using wire screens, mosquito nets or repellents. **4-**Vaccination in endemic areas using suspension of living promastigote resulting in lasting immunity.						

Parasite (Disease)	Toxoplasma gondii (Toxoploasmosis)
Geographical Distribution	Worldwide distribution
DH & Habitat	Cat
I.M. host	Wide range of avian and mammalian intermediate hosts including man and cats (Obligate Intracellular)
Diagnostic Stage	*Toxoplasma* trophozoites
Infective Stages & Mode Of Infection	Tachyzoite (trophozoite), bradyzoite, tissue cyst, pseudocyst and sporulated oocyst except for Unsporulated oocyst. Infection can occur through: ***Oral route***: most common route of infection , includes: • Ingestion of sporulated oocyst in contaminating food, drinks, objects, hands, etc… • Ingestion of cysts present in undercooked meat of intermediate hosts. ***Parenteral route***: • Trans-placental transmission: pregnant woman with parasitaemia. This can occur during: ○ Acute primary infection of pregnant woman during or shortly after pregnancy. ○ Old latent infection when the woman is immunosuppressed. • Blood and leukocyte transfusions if the donor has parasitaemia. • Organ transplantation, particularly heart transplants. • Contamination of mucous membranes and skin abrasions.
Life Cycle	• Asexual cycle (in the intermediate hosts; man, cattle, goats, poultry & cats). • Sexual cycle (in the definitive host; cats only).
Pathogenesis	**1.** Congenital infection: At first, there is generalized infection of the fetus then the parasite localizes in the CNS. Ocular lesions start by proliferation of the parasite in the retina and cause inflammation of the choroid (Retinochoroiditits). **2.** Acquired infection: ▪ In acute stage: focal areas of inflammation and necrosis in various tissues. ▪ In chronic or latent stage: subsidence of inflammation and formation of inactive cysts (with development of immunity in immuno-competent hosts). **3.** Recrudescence: during immunosuppression, cysts reactivated resulting in flaring up and possibility for disseminated infection.
Clinical Picture	Toxoplasmosis is asymptomatic in the great majority of cases, the sequelae of infection depend on: ▪ Infected person's immunity and age. ▪ Virulence of the infecting strain of the parasite (possibly). ***I. Congenital toxoplasmosis***: The effect depends on age of the fetus at the time of infection and the protective immunity of the mother. Higher severity of infection occurs with early pregnancy infection. **1.** Loss of fetus due to abortion or still birth. **2.** Early neonatal manifestations in the form of: ▪ CNS affection: microcephaly, hydrocephalus, spasticity, palsy and convulsions. Cerebral calcifications could be seen on X-ray examination. ▪ Eye affection: retinochoroiditis (the most common sequelae of toxoplasmosis). ▪ Systemic manifestations: as fever, pneumonitis, hepatomegaly, jaundice and lymphadenitis. **3.** Late manifestations: infected baby appears healthy, manifestations appears late in life. - CNS involvement including mental retardation. - Eye affection. ***II. Acquired toxoplasmosis :*** • Lymphadenitis is the most clinical form of infection. It may be associated with fever, headache, myalgia and sometimes splenomegaly and skin rash. • Retinochoroiditis: it may be due to congenital infection that did not detected early. It can result in blindness. ***III. Toxoplasmosis in immuno-compromised patients (Opportunistic infection)*** : • Encephalitis: it is the most important manifestation in immuno-compromised patient and a major cause of death in AIDS patients. It is usually due to reactivation of latent cerebral cysts. • Organ transplant patients can develop acute disseminated toxoplasmosis.
Diagnosis	**Clinical**: suggestive as above **Imaging**: ▪ X-ray reveals cerebral lesions. ▪ Ultrasound, fetal examination can detect lesions as enlargement of cerebral ventricles. **Laboratory diagnosis**: • ***Serology***: Diagnosis mostly relies on serology. ➢ Detection of IgM is important as it indicates: ▪ Active infection ▪ Congenital infection as the maternal IgM does not cross the placental barrier. ➢ Detection of IgG indicates: ▪ Acute infection (rising titre): appears later than IgM and usually persists for a year. ▪ Chronic latent infection: stable or declining titre. ➢ Techniques: various as ELISA, IFT, etc… ➢ Sabin Feldman dye test: serum of patient is added to Toxoplasma organisms and methylene blue. If antibodies are present, the organism will not take the dye → positive reaction. • ***Frenkle test (Toxoplasmin intradermal test)***: positive in active and chronic infections and has limited clinical applications. • ***Molecular techniques***: PCR is especially important in immuno-compromised patients in whom immuno-diagnosis is not reliable. Also, it can be used on amniotic fluid samples and infant urine for diagnosis of congenital infection.
Treatment	• ***Combination of*** Pyrimethamine and Trisulphapyrimidines. • Spiramycin can be given for infected pregnant women.
Prevention & Control	**1.** Proper washing of hands, vegetables and fruits, clean water supply and safe disposal of cat's litter boxes. **2.** Washing of hands and utensils after handling raw meat & proper freezing and cooking of meat. **3.** Health education of pregnant women and routine antenatal serological screening to detect maternal infection.

Parasite (Disease)	Potentially pathogenic free-living amoeba			Microsporidia
	Naegleria fowleri (Primary Amoebic Meningioencephalitis; PAM)	Acanthamoeba species		
		Granulomatous Amoebic Encephalitis (GAE)	Acanthamoeba Keratitis	
Geographical Distribution	Reported in some parts of the worlds	Sporadically reported		Reported from various parts of the world.
Habitat in Nature	Water (fresh, brackish & salt), moist soil, decaying vegetation.			----------------
Diagnostic Stage	Amoeboid trophozoite form	trophozoite form	Trophozoites & cysts	Spore stage
Infective Stage & Mode Of Infection	Amoeboid trophozoite, through **nasal route** during swimming in or sniffing of contaminated water & inhalation of contaminated air.	Trophozoite, through: contaminated water & air: 1ry infection → nasal route 2ry infection → blood spread	Amoeba, through: • Corneal trauma. • Exposure to contaminated water. • Wearing contaminated contact lenses.	The spores, through uncertain methods but the infection is most likely acquired by ingestion. Others include inhalation, ocular exposure & sexual intercourse.
Life Cycle	Flagellate form ↔ Amoeboid form ↔ cyst stage	Trophozoite (active) ↔ Cyst (resting)		Spores → Ejects the tubular polar filaments → Sporoplasm → Cytoplasm of the host cell → Cycles of Merogony followed by Sporogony → Spores
Pathogenesis	**1.** Amoeboid trophozoite invades the nasal mucosa and cribriform plate and reaches the brain along the olfactory nerves. **2.** Flagellate & cyst give rise to Amoebae prior of invasion, which is the only form detectable in brain tissue. **3.** Naeglaria produces diffuse **meningio-encephalitis** with hemorrhagic inflammation and necrosis of brain tissue.	**1.** 1ry infection occurs in the lower respiratory tract and ulcerated skin & mucosa. **2.** Invasion of CNS (2ry infection) by blood spread causes single or multiple focal granulomatous lesions in the brain & other affected organs. **3.** In AIDS patients disseminated infection can developed.	**1.** Acanthamoeba cause chronic progressive ulcerative keratitis. Corneal ulceration may progress to perforation. **2.** In AIDS patients, infection may cause endophthalmitis. **3.** The infection is characterized by sever ocular pain and affection of vision.	It depends on the type of the microsporidia: ***Intestinal microsporodiosis***: The most clinical form & is usually seen in AIDS patients. It produces prolonged diarrhea, malabsorption, wasting and dehydration. Cholangitis and rhinosinusitis can develop due to spread to other epithelial cells. Systemic disease can also develop due to spread to multiple organs. ***Ocular microsporidiasis:*** Ocular lesions affect both healthy and HIV infected subjects. Infection can lead to conjunctivitis, keratitis and corneal ulcers. ***Microsporidial myositis:*** The patient suffers from generalized muscle weakness, myalgia, fever and weight loss. ***Systemic infection:*** Infection can involve several systems in both immunocompetent and immunocompromized patients. There may be intestinal, biliary, ocular, hepatic, renal and respiratory affections.
Clinical Picture	Pam is an acute fulminant rapidly fatal disease that affects mostly children and young adults **1.** There is fever, headache, nausea & vomiting, stiffness of neck and convulsions. **2.** Disturbance in the sense of smell or taste can occur. **3.** The patient enters in coma and death occurs early. **4.** The entire course usually takes 3 – 6 days.	Takes subacute or chronic course (days to years) • Manifested by nausea & vomiting, altered mental state, headache, convulsions and stiffness of the neck. • In AIDS patients, the disease may be fulminating resembling Naeglaria infection.		
Diagnosis	**1.** History of swimming mainly. **2.** CSF examination: ▪ Microscopic examination reveals amoeba forms. ▪ Suspension in fresh water incites transformation into flagellate forms that confirm the diagnosis. ▪ Culture on suitable medium	CSF examinationreveals the parasite.	Identification of trophozoites & cysts in corneal scraping directly and after culture.	***Direct methods:*** • Biopsy: identification of organisms in stained biopsy material. • Examination of excreta and body fluids: identification of stained spores in faeces, urine, bile and duodenal, bronchial or nasal fluisds. • Electron microscopy: to identify the ultra-structure of the parasite. ***Molecular techniques:*** Assays are being developed.
Treatment	**1.** At present, there is no complete treatment. **2.** Amphotericin B can be given IV or intrathecally.	There is no complete satisfactory treatment. However, there are some re-ported successful regimens: • Excision of focal lesion & treatment with Ketoconazole. • Penicillin & Chloramphenicol	• Oral Itraconazole ***combined with*** topical Miconazole. • Corneal transplant.	Albendazole: for intestinal and disseminated infections. Nitazoxanide: is effective in intestinal microsporidiosis. Topical fumagillin: in ocular lesions.
Prevention & Control	**1.** Avoidance of swimming in contaminated water. **2.** Proper chlorination of public water supplies & pools.		**1.** Proper care of contact lenses. **2.** Avoidance of exposure of the eye to contaminated water.	----------------

Commensal amoebae

They are characterized by:

1- All move by pseudopodia, giving rise to a sluggish hesitant motility.
2- Endoplasm is not clearly differentiated from the ectoplasm with food vacuole containing bacteria and tissue debris showing a dirty endoplasm (contrary to E. histolytica)

Etnamoeba dispar:
It is morphologically similar to E. histolytica but differs in being non-invasive living in lumen of large intestine.

Etnamoeba hartmanni:
It is morphologically similar to E. histolytica and only differs in size:

1- Trophozoite: range from 4 – 12 um in diameter and ingest bacteria only.
2- Cysts: range from 3 – 10 um in diameter.
3- It is non-pathogenic (not tissue invader).

Etnamoeba coli:
It lives in the large intestine in 10 – 30 % of people. It resembles E. histolytica but differs in:

1- Average size of trophozoite is larger being 30 um.
2- More granular endoplasm containing ingested bacteria but no red cells.
3- Narrower and less differentiated ectoplasm.
4- Broader and blunter pseudopodia.
5- Sluggish movement.
6- Peripheral chromatin granules of the nucleus are more coarse irregular.
7- Karyosome is large and eccentric.
8- Cysts are larger (average size is 25 um) with slender aplinter-chromotoid bodies, glycogen vacuole and 8 nuclei similar to those of the trophozoite stage.

N.B. Enough to study the names of commensal amoebae & know that E. coli is bigger, E. hartmanni is smaller & E. dispar is morphologically identical to E. histolytica

Immunology of Parasitic Infections

Vaccination

No efficient vaccine preparations have yet been approved for human use against parasites. The causes include:

1. Difficulty of identification and isolation of protective antigens to be used as vaccine.
2. Possibility of induction of immunopathological lesions in response to the vaccine.
3. Parasites may evade the immunity produced by the vaccine.
4. The complexity of the life cycles of the parasites makes the choice of the stage to be targeted difficult.
5. Vaccine preparations may not be equally effective in different countries. This is due to the existence of various strains for the same parasite in different geographical regions.

Immunopathology of parasitic infections

1. Allergic reactions (Type 1 hypersensitivity): IgE mediated allergy commonly occur with helminthic infections. They include:
   - Systemic reactions (anaphylaxis): life-threatening condition, e.g. after leakage of hydatid cyst fluid.
   - Bronchial asthma: e.g. Fascioliasis and Larva migrans.
   - Allergic dermatitis: e.g. early stages of Schistosomiasis, Fascioliasis and Ascariasis.
2. Eosinophilic pneumonia: results from helminthic larvae, e.g. Ascaris & Hookworms.
3. Autoimmune reactions: They are due to cross-reaction with the host antigens. They can lead to hemolytic anemia as in falciparum malaria and produce damage to the heart and neuronal tissue in Chagas' disease.
4. Deposition of immune complexes (Type 3 hypersensitivity): may deposited systemically or on basement membranes.
   e.g. Plasmodium malariae → Nephrotic syndrome
   Schistosomiasis →Katayama fever (serum sickness-like disease).
5. Granuloma formation (Delayed –type hypersensitivity): e.g. in Schistosomiasis, granuloma formation around the eggs.
6. Immunosuppression: Variable degrees of immunosuppression is observed in parasitic infections. e.g. infants infected with visceral Leishmaniasis may suffer severe immunosuppression. Death of these patients often results from secondary infections (e.g. pneumonia).

Parasite Immune Evasion

1. Sequestration of the parasite: It means the hiding of the parasite. It can be achieved through:
   - Intracellular habitat; e.g. Plasmodium & Toxoplasma.
   - Presence of surrounding cyst wall; e.g. Trichinella.
2. Luminal habitat: e.g. Ascaris and Enterobius.
3. Parasite movement: .e.g. Ancylostoma, Larva migrans.
4. Antigen modification: through:
   - Antigen variation: e.g. African trypanosomes, malaria.
   - Antigen disguise: Parasites cover themselves with various host proteins; e.g. Schistosomiasis.
   - Antigen mimicry: Presence of similarity between host and parasite; e.g. Schistosomiasis.
   - Antigen shedding: Parasites shed their surface antigens to neutralize the antibodies at a distance from the parasite; e.g. S. mansoni & P. falciparum.
5. Production of blocking antibodies: These are antibodies of little protective effect. They combine with helminth antigens making them unavailable for antibodies of high protective effect, e.g. Schistosomiasis.
6. Inhibition of immune factors:
   - Cleavage of antibodies: by protease enzyme, e.g. Trypanosoma cruzi
   - Inactivation of complement:
     - Protease activity: e.g. Schistosoma larva
     - Acceleration of decay of complement: e.g. Trypanosoma cruzi
     - Ejection of membrane attack complexes from their surface: e.g. Leishmania
   - Inhibition of macrophages: e.g. Leishmania, Toxoplasma & Trypanosoma cruzi
7. Immunosuppression e.g. visceral Leishmaniasis.

Immunodiagnosis of Parasitic Infections

- Situations where immunodiagnosis is important:
  1. When we cannot precisely locate the parasite e.g. visceral larva migrans.
  2. When parasitic stages may not appear in patient's excreta e.g. early and chronic phases of infection.
  3. To differentiate true from spurious (false) infections e.g. Fascioliasis.
  4. When large numbers of specimens are simultaneously tested e.g. epidemiological studies.
  5. When sampling may be dangerous to the patient e.g. hydatidosis, cerebral toxoplasmosis, trichinosis.
  6. Follow up after treatment.
- Serology is better than skin testing.
- Detection of antigen is superior to detection of antibody.

Progress in Molecular Parasitology

Molecular techniques are now being progressively applied in parasitology especially in these fields:

- Molecular classification of parasites:
  1. Morphologically similar parasites can be classified into species, subspecies, etc… according to DNA & RNA sequences.
  2. Molecular analysis can identify evolutionary relationship between parasites. The more similar the sequences in two organisms, the more likely they are related.
- Study of parasite biology:
  Such as metabolism, host invasiveness, virulence factors, etc…
- Development of new drugs:
  1. Identification of vital targets in the parasite that are lacking in the host allows the development of more effective and less toxic drugs.
  2. Detection of the drug resistance genes and understanding their mechanism of action permits the development of drugs that overcome this resistance.
- Diagnostics:
  1. Molecular diagnosis as PCR and DNA hybridization techniques.
  2. Molecular methods can be used to prepare sensitive and specific reagents to be used in immunodiagnosis.
- Vaccine development.
- Epidemiology and control measures:
  Determination of geographical distribution of various strains of the parasite allows the implementations of control measures most suitable for local strains.

Medical Entomology

Medical importance of arthropods:

I. ***Arthropods as agents of diseases and discomfort***:
   a. Inoculation of poisons: e.g. ticks, spiders & scorpions.
   b. Invasion of tissues: e.g. scabies & myiasis.
   c. Dermatitis and allergic skin lesions: e.g. fleas, mosquitos, bugs & lice.

II. ***Arthropods as transmitters of diseases***:
   a. Mechanical transmission:
      i. Indirect: They act as passive carriers of organisms on their hairs, mouth-parts or legs. e.g. Musca domestica.
      ii. Direct: They pick the organisms from a diseased person and inoculate them to healthy one. e.g. stomoxys.
   b. Biological transmission:
      i. Propagative: multiplication of the organisms without developmental changes. e.g. yellow fever virus (Aedes aegypti) & plague organisms (fleas).
      ii. Cyclopropagative: multiplication and developmental changes of the organisms. e.g. malaria (Anopheles) & Leishmania (Sand fly).
      iii. Cyclodevelopmental: developmental changes without multiplication. e.g. filaria (mosquitos).
      iv. Transovarian: organisms transmitted from the infected mother to offspring through the ova. e.g. organisms transmitted by hard & soft ticks.
      v. Trans-stadial (stage to stage): e.g. organisms of scrub typhus which pass from larvae (as ectoparasite of man) to nymph to adults to next larvae.

Arthropod	Geographical Distribution	Medical Importance	Mechanism of Transmission	Control (Read only)
Culex	Cosmopolitan but more prevalent in tropical and temperate countries.	Transmission of: • Wuchereria bancrofti (chief transmitter). • Viral encephalitis. • Rift valley fever (viral disease)	Transmission is only by females, which bites man sucking his blood.	***Aquatic stages (larvae & pupae):*** **a.** Mechanical (physical) control: • Removal of plants where larvae breed in shaded areas and development of shade when they need sunshine. • Filling or drainage of breeding places. • Production of turbidity in clear water. • Changing the current and the level of water to prevent permanent growth. **b.** Biological control: • Natural enemies such as frogs, ducks and fish. **c.** Chemical control: • Malariol (larvae & pupae): cheap non-volatile oil, which cause suffocation of aquatic stages. • Paris green (larvae, not pupae): a spray for the surface of water. It acts as stomach poison to larvae. It does not affect pupa as it does not feed. • Insecticides (DDT): produce poisoning whether ingested or contact the cuticle. ***Adult stages:*** • Wire screening. • Mosquito repellents. • Spraying with insecticides. • Animal barriers. • Sterilization of males by irradiation produces infertile eggs. • Using of natural enemies.
Aedes		Transmission of: • Yellow fever virus. • Dengue fever virus. • Wuchereria bancrofti. • Rift valley fever.		
Anopheles		Transmission of: • Human malaria. • Wuchereria bancrofti & B. malayi. • Viral encephalitis (occasionally).		
Phlebotomus (Sand Fly)	Prevalent in Mediterranean coasts, Middle east, Africa, India China and America. **N.B.** Phlebotoms papatasii is present in Egypt	**1.** Transmission of diseases: **a.** Protozoal (Leishmaniasis) **b.** Bacterial (Oroya fever = Carrion's disease = Bartonellosis) **c.** Viral (Sand fly fever = Papatasii fever = 3 day fever): similar to influenza. **2.** Harrara: allergic reaction to the bite of sand fly.	Transmission is only by female bite, feed by night and hide by day time.	**1.** Filling the cracks in walls and ground to deprive the fly from its breeding places. **2.** Screening of windows and doors by nets with narrow meshes. **3.** Insecticides against larvae and adults. **4.** Repellents to the skin.
Musca domestica (House Fly)	Cosmoplitan	It is considered as efficient disease agent transmitter: **1.** ***Indirect*** mechanical transmission of microorganisms (Typhoid, Poliomye-litis and bacillary dysentery), cysts of protozoa and eggs of helminthes. **2.** Accidental myiasis.	The tiny hairs covering the body, the mouth-parts and the legs have sticky pads, all help to collect organisms and transmitting them.	**1.** Elimination of breeding places. **2.** Spraying with insecticides (DTT). **3.** Wire screening of inlets and outlets of the house. **4.** Basic sanitation and health education (Musca develops resistance to ordinary insecticides).
Stomoxyscal-citrans (Stalble fly)	Cosmoplitan	**1.** ***Direct*** mechanical transmission of blood parasites as trypanosomes. **2.** Accidental myiasis. **3.** Painful bites. **4.** Skin allergy.	Direct mechanical transmission of blood and painful bites.	As musca but mainly to animal stables.
Glossina (Tse-Tse fly)	G. palpalis: West Africa G. morsitans: East Africa	**1.** Transmission of trypanosoma that cause sleeping sickness in man. **2.** Transmission of Nagana to animals.	By biting of tsetse fly	**1.** Changing the nature of breeding places to become unsuitable for the fly and periodic cleaning of riverine vegetation (deforestation). **2.** Collection of larvae and pupae. **3.** Application of insecticides. **4.** Treatment of patients. **5.** Campaign against wild animals.
Calliphorinae (Calliphora, Lucilia) **Sarcophaginae (Sarcophaga, Wohlfahrtia)**	Cosmopolitan	They cause ***semi-specific myiasis***	By sucking mouth	As Musca
Cimicidae (Bed Bugs)	Cosmopolitan, the commonest one in man is Cimex lectularius	**1.** Their persistent biting by night causes insomnia and nervous irritability. **2.** They may act as mechanical carriers but they are not biological vectors of human diseases. **3.** Recently, there is evidence to indicate that they may transmit hepatitis B virus.		**1.** Application of insecticides to hiding places. **2.** Fumigation with sulfur. **3.** Collection and destruction of bugs.
Reduviidae (Winged Bug)	North & South America	▪ Also called: winged bug, cone-nosed, kissing, Assassin & Barber's bug. ▪ They are the vectors for T. cruzi that causes Chagas' disease and T. rangeli which is non-pathogenic to man.		As bed bugs.
Cyclops	Live in fresh water.	Act as Intermediate host for: D. latum, D. mansoni & D. medinensis.		• Regular steaming or addition of calcium oxide, chlorine or copper sulphate. • Fish (Barbus): can feed on Cyclops. • Wells water should be boiled, filtered, covered and provided with pumps.

Arthropod	Geographical Distribution	Medical Importance	Mechanism of Transmission	Control
Siphonaptera (Fleas)	Cosmopolitan	***As vector of diseases:*** **1.** Plague: (causative organism is Yersinia bacilli) Ingestion of infected blood → flea's stomach → multiplication & block of the stomach → next ingestion of blood → blood cannot pass the obstruction →regurgetation with bacilli (Anterior station transmission) **2.** Endemic or murine typhus: (causative organism is Rickettsia mooseri; R. typhi): Rickettsia →epithelial lining of mid-gut → multiplication → faeces (Posterior station transmission) *Man is infected trough*: **a.** Contamination of skin by flea faeces or by scratching. **b.** Inhalation of dried flea faeces (bacilli still alive for 40 days). **c.** Crushing the fleas. ***As intermediate host of parasitic diseases:*** 1. Rat fleas act as IH for H. nana & H. diminuta. 2. Dog and cat fleas act as IH for D. caninum. ***Fleas attacking their host:*** Tunga penetrans causing Chigger's or Jigger's disease. ➢ It is found in tropical & subtropical regions. ➢ The fertilized female burrow into the skin of the sole of foot or between the toes to take its blood meal. ➢ As eggs develop they project to live in the soil. ➢ Clinical picture: painful nodular swelling which may ulcerate. ➢ Treatment: surgical removal of flea with antiseptic dressing & antibiotics. ➢ Prevented by wearing shoes.		***Human fleas***: **1.** Application of insecticides under the carpets. **2.** Use of vacuum cleaners ***Dog and cat fleas***: Dusting animals and their homes with insecticides. ***Rat fleas***: **1.** Dusting rat holes with insecticides. **2.** Using of rodenticides (warfarin). **3.** Strict quarantine measures against ships coming from foreign parts by fumigation to kill rats.
Anoplura (Lice)	Worldwide, wherever there is low hygiene	***Lice as vectors of diseases (Body lice):*** **1. *Epidemic typhus***: causative organism is Rickettsia prowazekii. ➢ Mechanism of transmission: Rickettsia → lice gut cells → multiplication → rupture of cells → lumen → pass with faeces ➢ Infection occurs by: ▪ Contamination of bite wound with lice faeces (posterior station) ▪ Inhalation of dust containing the dried infected faeces of lice. ▪ Crushing the lice against skin abrasions. ➢ The source of the epidemic infection is either: ▪ A case of typhus from neighboring area. ▪ A case of Brill-Zinsser disease: it is a mild form of typhus. It is a late recrudescence of long dormant infection (may be 30 years of 1st infection). **2. *Trench fever (5-day fever):*** causative organism is Rickettsia Quintana. ➢ Mechanism of transmission: ▪ Contamination of skin wound with lice faeces (posterior station). ▪ Crushing the lice against skin abrasions. **3. *Epidemic relapsing fever:*** causative organism is Borrelia recurrentis. ➢ Mechanism of transmission: **Only** by crushing the lice against the skin. ***Lice as a cause of Pediculosis (Vagabond's disease):*** It occurs in persons who have lice for long periods. The skin becomes thickened and shows spots of hyperpigmentation. ***Pubic louse (Phthirus pubis):*** **1.** It is not known to transmit any disease. **2.** It causes irritation of the skin which shows bluish patches. **3.** If present in the eyelashes, it causes inflammation of lid margin (blepharitis).		***Body lice (Pidiculus humanus corporis)***: Frequent bathing and boiling of clothes. ***Head lice (Pidiculus humanus capitis)***: The current drugs of choice are: ▪ Synthetic pyrethrin as a spray. ▪ Anticholine esterase inhibitors. ***Pubic lice***: ▪ Boil the underwear. ▪ Shave the pubic and axillary hairs. ▪ Removal of the lice from eyelashes with forceps and application of yellow oxide of mercury ointment .
Acarina (Ticks)		***Diseases caused by ticks:*** **1.** Dermatosis: during biting, they produce trauma to the skin by the mouth-part. This provokes inflammatory reaction. Forcible removal of the tick may be complicated by 2ry infection and ulceration. **2.** Paralysis: a rapid ascending flaccid paralysis with difficulty in swallowing and respiration that may lead to death especially in children & aged adults. It caused by toxins in their saliva. Most of the patients recover after removal of the tick. ***Diseases transmitted by hard ticks:*** ➢ Rickettsial diseases:as Q-fever by Coxiella burneti. ➢ Bacterial & spirochaetal diseases: as Lyme disease by Borrelia burgdorferi. It is a systemic illness with skin lesions, fever, arthritis, carditis or meningitis. ➢ Viral disease: as viral encephalitis & Haemorrhagic fever. ➢ Protozoal diseases: Babesiosis by Babesia divergens. ***Diseases transmitted by soft ticks:*** Endemic relapsing fever by Borrelia duttoni. Q-fever by Coxiella burnetti .	Occurs in hard ticks either by: • Saliva in the bite wound • Contamination of skin abrasions by faeces. • Trans-ovarian transmission. Occurs in soft ticks either by: • Saliva • Coxal fluid • Trans-ovarian transmission	**1.** Careful search for ticks in persons exposed to infected areas and early removal of ticks by ***gentle extraction*** after applying chloroform, ether, kerosene or a glowing match or cigarette to the tick avoiding breaking down the capitulum. **2.** Soft ticks are killed by spraying their hiding places with insecticides. **3.** Hard ticks being permanent ecto-parasites, insecticides should be applied directly by spray or dipping the domestic animals in basins containing 5% gammaxane. **4.** Using repellents, wearing high boots and clothes treated with diethyltoluamid. **5.** Rodent proofing buildings. **6.** Anti-tick vaccine is proved to effective in veterinary practice.

Arthropod	Geographical Distribution	Pathogenesis (Medical Importance) & Clinical Picture	Diagnosis	Treatment	Control
Sarcoptes scabiei & Scabies (Itch mite)	Worldwide, especially among poor classes or when there is over crowding or lack lf hygiene.	***Common sites:*** Inter-digital spaces, flexor aspects of wrist and forearm, elbow, axillae, back, inguinal region and genitalia. ***The lesions:*** **1.** Elevated reddish tracks in the skin with minute vesicles. **2.** The patient suffers from intense itching, which is aggravated by warmth and sweating causing scratching. **3.** This spreads the lesions and induces 2ry bacterial infection. **4.** This results in multiple popular vesicular & pustular lesions with widespread eruptions. ***Crusted or Norwegian scabies:*** **1.** This is a generalized dermatitis with extensive scaling and crusting. **2.** It may occur in immuno-deficient or very debilitated patients with hundreds of mites in the lesion.	**1.** Clinical picture: previously. **2.** Examining the skin surface with a hand lens to find the burrows & opening one of them by a needle to see the mites. **3.** Scraping the infected area with a scalpel and material obtained is examined microscopically, immediately after adding 10% KOH to avoid dissolving of the mites. **4.** A better method is by applying mineral oil to the skin before scraping. This enables organisms to adhere better to the blade and the slide and will not dissolve the mites.	**1.** The patient must take hot soapy bath using a rough brush (to open the tunnels). **2.** Many acaricides are used, the most effective are: **a.** Pyrethrin as 5% cream. **b.** Crotamiton (Eurax) 10% is preferred in infants, children and pregnant women. **c.** Lindane 1% cream. **d.** Benzyl benzoate 20%. The drug must be applied to the whole body for 8 – 12 hours then washed off. **3.** Antihistaminics for itching and antibodies for 2ry infections.	**1.** Frequent bathing and boiling of bed linen. **2.** Avoid contact with patients and infected animals. **3.** Treatment of patients and domestic animals.
Demodex folliculorum (Follicle mite)	Cosmopolitan	**1.** They rarely cause any harm but the may be associated with acne and comedones (black heads). **2.** They may cause dry erythema with scaling and blepharitis. **3.** Infestations are usually higher in aged persons and in women using cleansing creams instead of soap and water.	Pressing the lesion and examining the extruded material microscopically for parasitic stages.	**1.** Washing the face with soap and water. **2.** Lindane 0.5% in vanishing cream combat them.	
Trombiculid mites	-------------	• They are called Chigger's mites, Harvest mites or Red bugs. • Vectors of scrub typhus: by trans-ovarian & trans-stadial transmission. • Chigger's mites causing dermatitis: by larvae in North America & Europe.	---------------------	---------------------	Personal protection by impregnation of socks and trousers with a repellent (pyrethrin or diethyl-tolumid). This will prevent attack of mites.
House dust mites	-------------	**1.** Bronchial asthma: especially in children. **2.** Perinnial rhinitis: sneezing, nasal congestion, watery discharge and conjunctival itching. **3.** Dermatitis: erythematous scaling & lichenified areas.	**1.** History taking. **2.** Clinical examination. **3.** Determine sensitization by skin tests and serum assays of specific IgE and IgG4 antibodies.	---------------------	**1.** Exclusion of dust from beds and bedrooms of patients. **2.** Indoor humidity control. **3.** Vacuum cleaning. **4.** D'allergen (acaricidal and allergen reducing agent).
Storage (Forage) mites	-------------	Pests of stored food products e.g. flour, cereals, cheese and macaroni. They affect workers handling these products (exposed to crushed mite products and their excreta) e.g. baker's itch or grocer's itch. **1.** Dermatosis: by contact or bite of mites.it produces itching, urticarial and papular eruptions of exposed parts of the body. **2.** Digestive troubles: if swallowed, they cause irritation of intestinal crypts with colic & they are recovered in faeces (intestinal acariasis). **3.** Respiratory symptoms: if inhaled, they are recovered in sputum (respiratory acariasis). **4.** Allergic conjunctivitis.	---------------------	• A lotion of worm water and vinegar or sat. solution of picric acid in 90% alcohol. • Saline purge in GIT troubles. • Arsenicals for respiratory symptoms.	
Domestic mites	-------------	• These are blood-sucking mites that cause dermatitis in man. • Rat mites are found in ware houses. Bird mite are found in air-conditioning ducts or eaves of houses.	---------------------	---------------------	---------------------
Scorpion	-------------	• Scorpion sting is very painful. • Its toxin causes twitching, muscle spasms, convulsions, shock & may cause heart failure, especially children & elders.	---------------------	**1.** Application of tourniquet just above the site of bite to decrease the absorption of the toxin. **2.** Suction of the venom or incision at the site of the wound. **3.** Doctors should be ready with anti-scorpion serum. **4.** Treatment of shock if present. **5.** Analgesics like aspirin, spraying anesthetics or corticosteroids in severe cases.	---------------------

Myiasis

- ***Definition***: It is the invasion of tissue of animals or humans by the larval stages of dipterous flies.
- ***Classification:***
  - **I. *According to habitat (type of tissue invaded)***:
    - **a.** *External myiasis*:
      - **i.** Cutaneous myiasis:
        - Invasion of intact skin: by larvae:
          - **a.** *Creeping eruption*: form tunnel under the skin e.g. Gastrophilus & hypoderma.
          - **b.** *Nodular swelling*: invade the intact skin & produce nodular swellings e.g. Cordylobia & Dermatobia.
        - Traumatic (wound) myiasis: invade wounds or ulcers e.g. Calliphora, Lucilia, Chrysomyia & Cochlyomyia.
      - **ii.** Nasal myiasis:
        e.g. Chrysomyia, Sarcophaga & Wohlfahrtia. Eggs → nasal cavity → hatch → larvae →burrow into tissue → bone → brain → meningitis & death.
        Clinical picture: nasal obstruction, sneezing & epistaxis.
      - **iii.** Ocular myiasis:
        - External ophthalmomyiasis (Oestrus):
          Adult Oestrus → eye → eggs → hatch → larvae which possess hooks→ conjunctival irritation & severe pain.
        - Internal ophthalmomyiasis (Oestrus, Gastrophilus, Hypoderma):
          It involves the orbit and eye. It is very destructive & leads to loss of eye.
      - **iv.** Aural myiasis:
        Severe pain accompanied by deafness & tinnitus and the drum can be perforated e.g. Chrysomyia, Sarcophaga & Wohlfahrtia.
    - **b.** *Internal myiasis*:
      - **i.** Intestinal myiasis:
        - Through ingestion of eggs or larvae in contaminated food, e.g. Musca, Calliphora & Sarcophaga.
        - Larvae deposited around the anus then reach the intestine, e.g. Fannia.
        - *Clinical picture*: abdominal discomfort, vomiting & diarrhea. Larvae may appear in the vomit or stool leading to patient's anxiety.
      - **ii.** Gastric myiasis:
        e.g. Eristalis, *Clinical picture*: vomiting.
      - **iii.** Urogenital myiasis:
        e.g. Fannia, *Clinical picture*: inflammation of urinary tract, pain during urination.
  - **II.** *According to the habit of the fly*:
    - **a.** ***Specific***: the larvae of this group are obligatory tissue parasites and can only develop on or in living tissue.
      - **i.** Dermatobia:
        - e.g. Dermatobia lays its eggs on mosquito when mosquito stands on human skin, eggs hatch..
        - The hatched larvae penetrate the skin and forms a nodule.
      - **ii.** Cordylobia:
        - e.g. Cordylobia.
        - Larvae are acquired from lying on the ground or from the clothes as the eggs are laid on contact to human skin, larvae come out of eggs & attack the skin.
      - **iii.** Oestrus, Gastrophilus and Hypoderma.
    - **b.** ***Semi-specific***:
      The larvae of this group usually grow on dead tissue of man and animals but they may invade neglected wounds. e.g. Calliphora, Lucilia, Sarcophaga & Wohlfahrtia.
    - **c.** ***Accidental***:
      Larvae may accidently get in the tissue when the eggs are ingested accidently with food. e.g. Musca & Piophila.
- ***Diagnosis***:
  Only by finding the larvae in the lesion & demonstrating its characteristic posterior spiracles.
- ***Treatment***:
  1. Removal of the larvae:
     - **a.** Manually if larvae are in skin, eye, nose and ear.
     - **b.** By saline purge if larvae are in stomach or intestine.
     - **c.** By douches if larvae are in vagina or bladder.
     - **d.** Through a cystoscope in urinary myiasis.
  2. Treatment of secondary infection (Antiseptics & Antibiotics).
- ***Prevention and Control***:
  1. Control of adult flies with insecticides and by the use of nets.
  2. Preventions of intestinal myiasis by protection of food from flies.
  3. Preventions of wound myiasis by cleaning and covering the wounds by gauze.

Serological tests are not necessary for the diagnosis of:

- ***Trematoda:***
  - Heterophyes
- ***Cestoda:***
  - Multiceps multiceps
  - Dipylidium Caninum
  - Diphyllobothrium latum
  - Diphyllobothrium mansoni & proliferum
  - Hymenolepis nana
  - Hymenolepis diminuta
- ***Nematoda:***
  - Enterobius vermicularis
  - Trichostrongylus colubriformis
  - Cutaneous larva migrans
- ***Protozoa, As regards the following Protozoa IT IS RECOMMENDED TO DO SEROLOGICAL TESTS IN THE FORM OF COPRO-ANTIGEN DETECTION using ELISA for example:***
  - Balantidium coli
  - Cryptosporidium parvum
  - Cyclospora cayetanensis
  - Isospora belli
- ***Artropods:***
  - All arthropods

Skin tests:

- ***Cestodes:***
  - Cysticercosis; Taenia solium (Taeniasis Solium)
  - Echinococcus granulosus (Hydatidosis, Hydatid disease)
  - Trichinella spiralis (Trichinosis)
  - Dracunculus medinensis (Dracunculiasis, Dracontiasis)
- ***Protozoa:***
  - Leishmania (Montenegro (leishmanin) test)
  - T. cruzi (Cruzin test)
  - Toxoplasma gondii (Frenkle (Toxoplasmin intradermal) test))
- ***Arthropods:***
  - House dust mites

Special tests:

1. Blood flukes (Schistosomiasis): Hatching test
2. Enterobius vermicularis (Enterobiasis):
   - **a.** N.I.H. swab (National Institute of health)
   - **b.** Scotch adhesive tape swab
3. Wuchereria bancrofti (Bancroftian filariasis, Elephantiasis): Di-ethyl-carbamazine (DEC) provocative test
4. Onchocerca volvulus (Onchocercosis or Onchocerciasis): Mazzotti test
5. Giardia lamblia (Giardiasis): String test (Enterotest)
6. Toxoplasma gondii (Toxoploasmosis): Sabin Feldman dye test

Surgical treatment:

- ***Cestodes:***
  - Diphyllobothrium mansoni & proliferum (Sparganosis)
  - Taenia solium (Cysticercosis)
  - Echinococcus granulosus (Hydatidosis, Hydatid disease)
  - Echinococcus multilocularis
  - Multiceps multiceps (Coenurosis)
- ***Nematodes:***
  - Wuchereria bancrofti (Bancroftian filariasis, Elephantiasis)
  - Loa loa: Eye African worm (Loaiasis or Loiasis)
  - Onchocerca volvulus (Onchocercosis or Onchocerciasis)
- ***Protozoa:***
  - Entamoeba histolytica (Amoebiasis; Cysts in tissues)
  - Cutaneous Leishmania (Cutaneous Leishmaniasis)

Biopsy (not absolute):

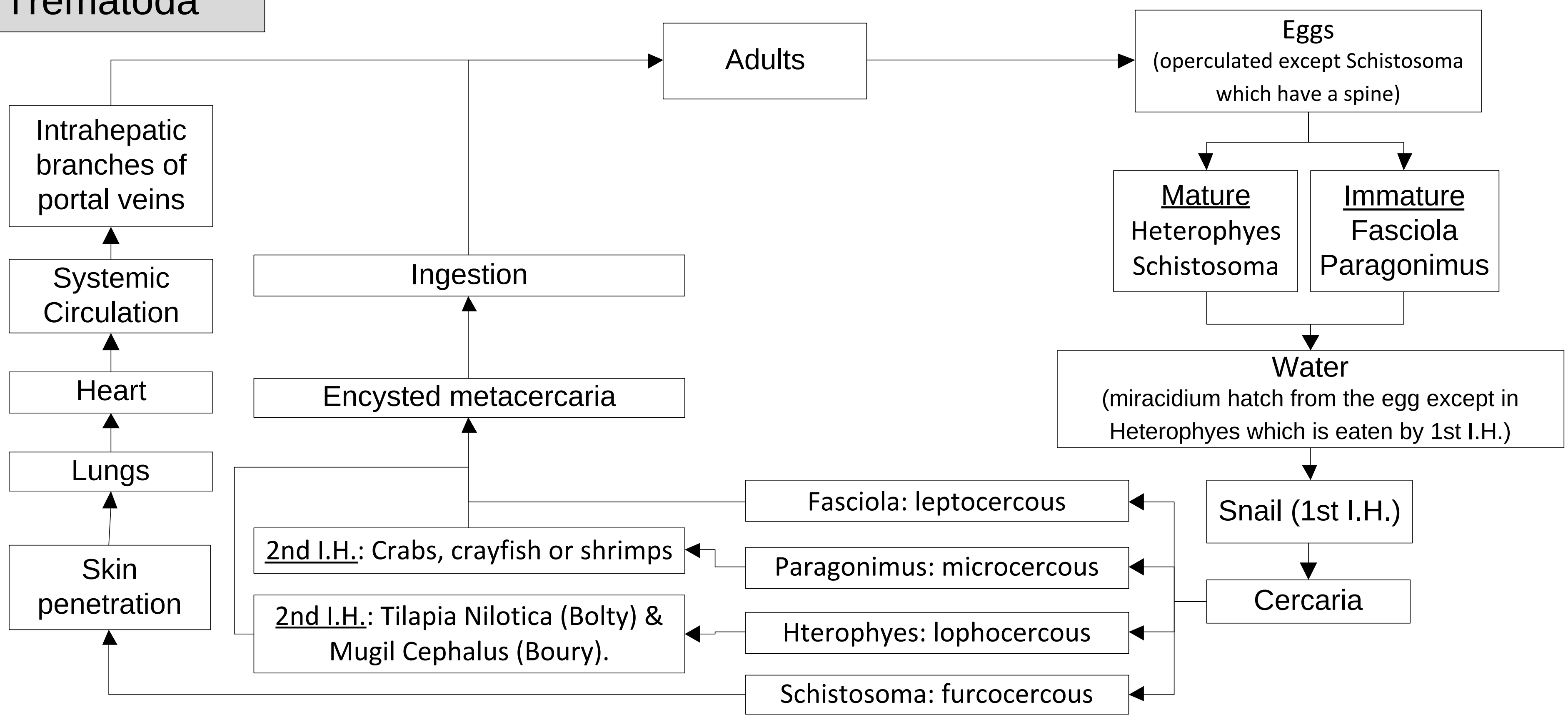
- ***Cestodes:***
  - Cysticercosis
- ***Nematodes:***
  - Visceral larva migrans (VLM)
  - Trichinella spiralis
  - Wuchereria bancrofti
  - Onchocerca volvulus
- ***Protozoa:***
  - Entamoeba histolytica
  - Cryptosporidium parvum
  - Cyclospora cayetanensis
  - Trypanosoma
  - Leishmania (Visceral & Cutaneous)
  - Microsporidia

Animal inoculation: • Trypanosoma

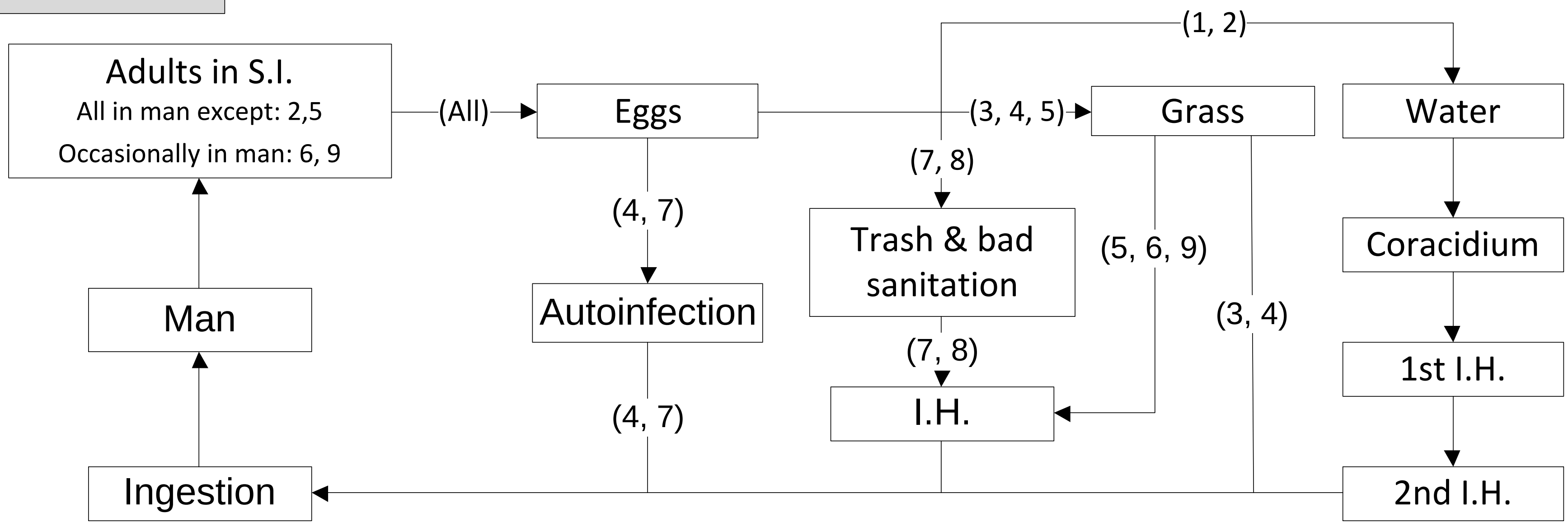
Life cycles of some Helminthes

2010 / 2011

Trematoda

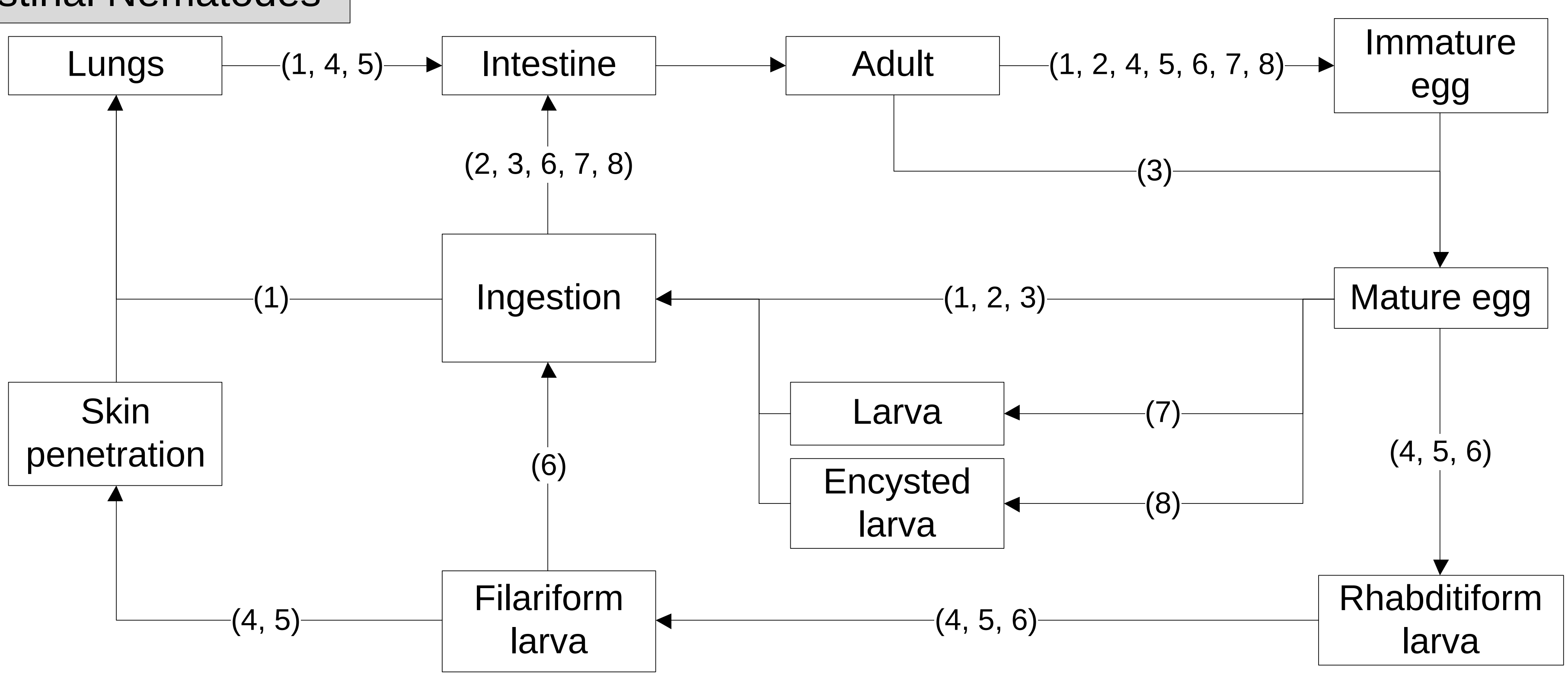


Cestoda



1: D. latum	2: D. mansoni & proliferum	3: T. saginata	4: T. solium	5: E. granulosus
6: M. Multiceps	7: H. nana	8: H. diminuta	9: D. caninum	

Intestinal Nematodes



1: Ascaris lumbricoides	2: Trichuris trichiura	3: Enterobius vermicularis	4: Ancylostoma duodenale
5: Strongyloides stercoralis	6: Trichostrongylus colubriformis	7: Capillaria philippinensis	8: Trichinella spiralis

Infective and diagnostic stages of Protozoa

Protozoon	Diagnostic stage	Infective stage
Entamoeba histolytica	Cyst or Trophozoite stage	Quadrinucleate (mature) cyst
Balantidium coli	Cyst or Trophozoite stage	Cyst stage
Giardia lamblia	Cyst or Trophozoite stage	Cyst stage
Cryptosporidium parvum	Oocyst stage	Both thin & thick-walled Sporulated oocyst
Cyclospora cayetanensis	Unsporulated oocyst stage	Mature sporulated oocyst
Isospora belli	Oocyst stage	Sporulated oocyst
Trichomonas vaginalis	Trophozoite only	Trophozoite stage
Visceral Leishmania	Amastigote in blood or biopsy and promastigote in culture.	Promastigote stage
African Trypanosoma	Multi form trypanosomes	Short stumpy metacyclic trypanosomes
American Trypanosoma	C-shaped trypanosomes	
Plasmodium	Ring, trophozoite, schizont, gametocyte stages in infected RBCs, but with P. falciparum ring & gametocyte stages only.	Sporozoite stage
Babesia	Merozoites	Sporozoites
Cutaneous Leishmania	Amastigote in biopsy and Promastigote in culture.	Promastigote form
Toxoplasma gondii	*Toxoplasma* trophozoites	Tachyzoite (trophozoite), bradyzoite, tissue cyst, pseudocyst and sporulated oocyst except for Unsporulated oocyst.
Naegleria fowleri	Amoeboid trophozoite	Amoeboid trophozoite
Acanthamoeba; Granulomatous Amoebic Encephalitis (GAE)	trophozoite form	Trophozoite
Acanthamoeba Keratitis	Trophozoites & cysts	Amoeba
Microsporidia	Spore stage	The spores

Diagnostic stages	
Oocyst	➢ C. parvum ➢ Cyclospora ➢ Isospora belli
Trophozoite	➢ T. vaginalis ➢ Plasmodium ➢ T. gondii ➢ Acanthamoeba
Cyst or Trophozoite	➢ E. histolytica ➢ B. coli ➢ Giardia lamblia ➢ Acanthamoeba Keratitis
Ring, trophozoite, schizont & gametocyte stages	➢ Plasmodium except P. falciparum
Ring & gametocyte stages *only*	➢ P. falciparum

Infective stages	
Cyst	➢ Entamoeba histolytica (Quadrinucleate (mature)) ➢ Balantidium coli ➢ Giardia lamblia
sporulated oocyst	➢ Cryptosporidium parvum ➢ Cyclospora cayetanensis (Mature) ➢ Isospora belli ➢ Toxoplasma gondii
Promastigote	➢ Visceral & Cutaneous Leishmania
Trophozoite	➢ Trichomonas vaginalis ➢ Toxoplasma gondii ➢ Naegleria fowleri (Amoeboid) ➢ Acanthamoeba; (GAE)
Short stumpy metacyclic trypanosomes	➢ Trypanosoma
Sporozoite	➢ Plasmodium ➢ Babesia
Amoeba	➢ Acanthamoeba Keratitis

Diagnostic and Infective Stages of Helminthes

Diagnostic				Infective		
Eggs		Larvae		Eggs		Larvae
Immature	Mature	Cestoda	Nematoda	Cestoda	Nematoda	
➢ Fasciola ➢ Paragonimus ➢ D. latum ➢ Ascaris lumbricoides ➢ Trichuris trichiura ➢ Trichostrongylus colubriformis ➢ Hookworms ➢ Capillaria philippinensis	➢ *Heterophyes* ➢ *Schistosoma* ➢ *Taenia* ➢ *Hymenolepis* ➢ *Dipylidium caninum* ➢ *Enterobius vermicularis*	➢ *D. mansoni* ➢ *Taenia* ➢ *Echinococcus* ➢ *M. multiceps*	➢ *Ascaris* ➢ *Capillaria* ➢ Visceral larva migrans ➢ *Strongyloides* ➢ *Trichinella* ➢ *Dracunculus* ➢ Filariae	➢ *Taenia* ➢ *Echinococcus* ➢ *M. multiceps* ➢ *H. nana*	➢ Embryonated *Ascaris lumbricoides* ➢ Embryonated *Trichuris trichiura* ➢ *Enterobius vermicularis*	➢ Encysted metacercaria: *Fasciola, Paragonimus, Heterophyes.* ➢ Cercaria: *Schistosoma.* ➢ Plerocercoid: *D. latum,* Spargana. ➢ Procercoid: Spargana. ➢ Cysticercus: *Taenia.* ➢ Cysticercoid: *H. nana, H. diminuta, D. caninum.* ➢ Filariform larva: Hookworms, Cutaneous larva migrans, *Strongyloides, Trichostrongylus.* ➢ Filiform larva: *Wuchereria bancrofti, Brugia malayi.* ➢ Infective larvae: *Loa loa, Onchocerca.* ➢ Encysted larva: *Trichinella.* ➢ Infective larva: *Capillaria, Dracunculus*

Cestoda		***Nematoda***			
Asis (adult, S.I.)	Osis (larva, tissues)	Oviparous (Does not require I.H.)		Larviparous (Requires I.H. or vector)	
Man act as D.H.	Man act as I.H.	Intestinal	Extra-intestinal	I.H.	Vector
➢ D. latum → Diphyllobothriasis ➢ Taenia → Taeniasis ➢ H. nana → Hymenolepiasis nana ➢ H. diminuta → Hymenolepiasis diminuta ➢ D. caninum → Dipylidiasis	➢ D. mansoni & proliferum → Sparganosis ➢ Taenia solium → Cysticercosis ➢ Echinococcus granulosus → Hydatidosis ➢ M. multiceps → Coenurosis	➢ Ascaris ➢ Trichuris ➢ Enterobius ➢ Trichostrongylus ➢ Hookworms ➢ Strongyloides	➢ Larva migrans (visceral & cutaneous)	➢ Capillaria ➢ Trichinella	➢ Dracunculus ➢ Filariae

Parasites affecting different organs

Organ	Trematoda	Cestoda	Nematoda	Protozoa	Arthropods
Small intestine	➢*H. heterophyes*	➢*D. latum* ➢*Taenia* ➢*H. nana* ➢*H. diminuta* ➢*D. caninum*	➢*Ascaris* ➢*Ancylostoma duodenale* ➢*Trichostrongylus* ➢*Strongyloides* ➢*Trichinella* ➢*Capillaria*	➢ *Giardia lamblia* ➢ *Cryptosporidium* ➢ *Isospora* ➢ *Cyclospora* ➢ *Microsporidia* ➢ Leishmania (in macrophage) ➢ Plasmodiunm (in blood vessels)	➢*Intestinal myiasis*
Large intestine	➢*Schistosoma mansoni*	-----------	➢*Enterobius* ➢*Trichuris*	➢ *Al amoebae except E. gingivalis* ➢ *Balantidium coli* ➢ Leishmania ➢ P. falciparum	➢*Intestinal myiasis*
Liver	➢*Fasciola* ➢*Schistosoma*	➢*Echinococcus granulosus* ➢*Cysticercus cellulosae*	➢*Ascaris* ➢Visceral larva migrans	➢ *Entamoeba histolytica* ➢ *Giardia lamblia* ➢ *Visceral Leishmania* ➢ *T. cruzi* ➢ *Plasmodium* ➢ *toxoplasma*	-----------
Lungs	➢*Schistosoma* ➢*Paragonimus*	➢*Echinococcus granulosus*	➢*Ascaris* (larva) ➢*Ancylostoma* (larva) ➢*Strongyloides* (larva) ➢Visceral larva migrans ➢*M. perstans* (pleura) ➢*M. ozzardi* (pleura) ➢*W.bancrofti*(tropical pulmonary eosinophilia)	➢ *Entamoeba histolytica*	-----------
Brain	Eggs act as emboli: ➢*Paragonimus* ➢*Schistosoma*	➢*Echinococcus granulosus* ➢*Cysticercus cellulosae* ➢Coenurus cyst	➢Visceral larva migrans ➢*Strongyloides* (disseminated larva)	➢*Entamoeba histolytica* ➢*Neglaria* ➢*Acanthamoeba* ➢*Trypanosoma* ➢*P. falciparum* ➢*Toxoplasma gondii*	-----------
Eye	-----------	➢Sparganum ➢*Cysticercus cellulosae*	➢Visceral larva migrans ➢*Trichinella* (larva) ➢*Loa loa* ➢*Onchocerca*	➢*Acanthamoeba (Keratitis)* ➢*T. cruzi (Romana's sign)* ➢*Toxoplasma gondii* (retinochoroiditis)	➢Ocular myiasis ➢*Phthirus pubis*
Heart	➢*Heterophyes*: eggs act as emboli	➢*Cysticercus cellulosae* ➢*Echinococcus granulosus*	➢Visceral larva migrans ➢*Trichinella (larva)* ➢*M. perstans* ➢*M. ozzardi*	➢*T. cruzi* amastigotes ➢*Toxoplasma gondii*	-----------
Lymph nodes	-----------	-----------	➢*Wuchereria bancrofti* ➢*Brugia malayi* ➢*O. volvulus*	➢*Trypanosoma* ➢*Leishmania* ➢*Toxoplasma gondii*	-----------
Skin & Subcutaneous tissue	-----------	➢Sparganum ➢Cysticercus cellulosae	➢Cutaneous larva migrans ➢*Dracunculus* ➢*Loa loa* ➢*Onchocerca*	➢*Leishmania* ➢African Trypanosome (chancre) ➢T. cruzi (Chagoma)	➢Cutaneous myiasis ➢*Sarcoptes scabiei* ➢*Tunga penetrans* ➢*Demodex folliculorum* ➢*House dust mites* ➢*Storage mites*
Muscles	-----------	➢Sparganum ➢*Cysticercus cellulosae*	➢*Trichinella*	➢*T. cruzi* ➢*Toxoplasma gondii*	-----------
Blood	-----------	-----------	➢*Wuchereria bancrofti* ➢*Brugia malayi* ➢*Loa loa* (microfilaria) ➢*M. perstans* ➢*M. ozzardi*	➢*Trypanosome* ➢*Leishmania* ➢Plasmodium ➢*Babesia*	-----------
Urine	➢*Schistosoma haematobium* egg	➢Hydatid sand from ruptured kidney cyst	➢*Enterobius* (in females) ➢Urogenital myiasis ➢*Wuchereria bancrofti* (microfilaria)	➢*Trichomonas vaginalis*	➢Urogenital myiasis
Sputum	Eggs of: ➢*Paragonimus* ➢*S. haematobium*	➢Hydatid sand from ruptured lung cyst	➢*Ascaris* (larva) ➢*Ancylostoma* (larva) ➢*Strongyloides* (larva)	➢*E. histolytica trophozoite* (from amoebic lung abscess eroding a bronchus) ➢*E. gingivalis trophozoite*	-----------

Modes of transmission of parasites

Mode	Trematoda	Cestoda	Nematoda	Protozoa	arthropods
Vegetables	➢*Fasciola*	➢*Taenia solium egg* ➢*H. nana* ➢*Echinococcus* ➢*M. multiceps*	➢*Ascaris* ➢*Enterobius* ➢*Trichuris* ➢*Toxocara (VLM)* ➢*Trichostrongylus*	➢*Amoebae* ➢*Giardia lamblia* ➢*B. coli* ➢*Toxoplasma gondii* ➢*Cryptosporidium* ➢*Isospora* ➢*Cyclospora*	➢Eggs of flies as Musca causing gastric or intestinal myiasis.
Water	➢*Fasciola* ➢*Schistosoma*	➢*Sparganum* ➢*Taenia solium* ➢*H. nana* ➢*Echinococcus* ➢*M. multiceps*	➢*Ascaris* ➢*Enterobius* ➢*Trichuris* ➢*Toxocara (VLM)* ➢*Trichostrongylus* ➢*D. medinensis*	➢Potentially pathogenic free-living amoebae	-----------
Undercooked fish	➢*Heterophyes* ➢*Paragonimus*	➢*D. latum*	➢*Capillaria*	-----------	-----------
Undercooked viscera/ muscles	➢*Fasciola* ➢*Linguatula* nymphs	➢Sparganum ➢*Taenia*	➢Trichinella spiralis	➢*Toxoplasma gondii*	-----------
Auto-infection	-----------	➢*H. nana* ➢*Taenia solium*	➢*Enterobius* ➢*Strongyloides* ➢*Capillaria*	➢*Entamoeba histolytica* ➢*Balantidium coli* ➢*Giardia lamblia* ➢*Cryptosporidium* ➢*Isospora*	-----------
Inhalation	-----------	-----------	➢*Enterobius*	➢Potentially pathogenic free-living amoebae	-----------
Congenital	-----------	-----------	-----------	➢*Toxoplasma gondii* ➢Plasmodium ➢*T. cruzi*	-----------
Through arthropods – Biological	-----------	➢*D. latum* ➢Sparganum ➢*H. nana* ➢*H. diminuta* ➢*D. caninum*	➢*D. medinensis* ➢All filaria	➢*Leishmania* ➢*Trypanosome* ➢*Plasmodium* ➢*Babesia*	-----------
Through arthropods – Mechanical	Eggs	-----------	-----------	Cysts	➢Blood parasites

❖ ***Geographical Distribution** (in Egypt or not?)*

- All Trematodes are present in Egypt except: *Paragonimus westermani*, *Schistosoma japonicum*.
- All Cestodes are present in Egypt except: *D. Latum* and *D. mansoni*.
- All intestinal Nematodes are present in Egypt except: *N. americanus*.
- The only tissue Nematode present in Egypt is *Wuchereria bancrofti*.
- All protozoa are present in Egypt except: L.donovani, L. chagasi, L. amazonensis, T. cruzi, P. ovale, P. malariae, P. falciparum, Babesia, L. aethopica & New World Cutaneous Leishmaniasis (NWCL).
- All arthropods are present in Egypt except: Glossina palpalis & Glossina morsitans & Reduviidae (Winged Bug), Trombiculid mite.

❖ ***Man as definitive host***

- In all Trematodes.
- In all Cestodes except: *E. granulosus*, *E. multilocularis*, *M. multiceps* and *D. mansoni*.
- In all intestinal nematodes except *A. caninum*, *A. braziliense*, *T. canis*, *T. cati*.
- In all tissue nematodes.

❖ ***Intermediate host***

- All Trematodes have one I.H. except: *Heterophyes*, *Paragonimus* have two I.H.
- All intestinal Cestodes have one I.H. except *D. latum* (two I.H.)
- Nematodes:
  - Intestinal nematodes: all have NO I.H. except: *T. spiralis*, *C. philippinensis*.
  - Tissue nematodes: all have I.H.

Special notes on Trematoda and Cestoda:

1- All are flat worms (Trematoda & Cestoda).
2- All are intestinal except Fasciola & schistosoma which are extra intestinal cestodes.
3- No gravid segment in Pseudophyllidea (D. latum & D. mansoni) because their uterus spell out eggs by its pores.
4- Any worm in small intestine ➔ nausea, diarrhea, vomiting & colic.
5- 4 worms have 2nd I.H.: Paragonimus (Crabs, crayfish or shrimps), Heterophys (Tilapia Nilotica (Bolty) & Mugil Cephalus (Boury)), Diphyllobothrium Latum (Salmon) and Diphyllobothrium Mansoni & Proliferum (frogs, snakes, mammals, birds, or man).
6- Intestinal obstruction ➔ constipation ➔ surgical treatment.
7- All eggs are yellowish brown except: Schistosoma, H. nana, *Enterobius, Ancylostoma, Trichostrongylus* (translucent).

Clinical presentations caused by helminthes **(VERY IMPORTANT TABLE)**

Disease	Trematoda	Cestoda	Nematoda	Protozoa	Arthropods
Diarrhea	➢*Heterophyes*	➢All adult Cestoda	➢Hook worms ➢*Ascaris* ➢*Strongyloides* ➢*Trichinella* ➢*Capillaria*	➢Giardia lamblia ➢Cryptosporium ➢Cyclospora ➢Isospora ➢Intestinal microsporidia ➢Visceral Leishmania ➢Malignant malaria (P. falciparum)	-----------
Dysentery	➢*Schistosoma mansoni*	-----------	➢*Trichuris*	➢*Entamoeba histolytica* ➢*Balantidium coli* ➢P. falciparum ➢Visceral Leishmania	-----------
Fever	➢*Fasciola* ➢*Schistosoma*	➢*Echinococcus granulosus (if ruptured into the blood)*	➢VLM ➢*Trichinella* ➢Filaria (bancrofti & malayi)	➢Amoebae ➢Potentially pathogenic amoeba ➢Trypanosome ➢Visceral Leishmania ➢Plasmodium ➢Babesia ➢Toxoplasma	-----------
Anemia – Microcytic hypochromic	➢*Schistosoma*	-----------	➢*Trichuris* ➢Hookworms	-----------	-----------
Anemia – Macrocytic hyperchromic	-----------	➢*D. latum* (vit. B12 deficiency)	➢*Trichuris* (due to toxic products)	-----------	-----------
Anemia – Normocytic	➢*Schistosoma*	-----------	-----------	➢Plasmodium ➢Babesia	-----------
Anemia – Hypoplastic	-----------	-----------	-----------	➢Leishmania ➢Trypanosome	-----------
Jaundice	➢*Fasciola* ➢*Schistosoma*	➢*Echinococcus granulosus*	➢Ascaris ➢Visceral larva migrans (VLM)	➢Amoebae ➢Plasmodium ➢Toxoplasma	-----------
Hepatomegaly	➢*Fasciola* ➢*Schistosoma*	➢*Echinococcus granulosus*	➢VLM	➢Amoebae ➢Visceral Leishmania ➢Trypanosome ➢Plasmodium ➢Toxoplasma	-----------
Splenomegaly	➢*Schistosoma*	-----------	-----------	➢Visceral Leishmania ➢Trypanosome ➢Plasmodium	-----------
Itching	➢Cercarial dermatitis	-----------	➢Hook worms ➢CLM ➢*Onchocerca* ➢*Loa loa*	-----------	➢Sarcoptes scabiei ➢Insect bites ➢House dust mites
Pruritis ani	-----------	➢*Taenia saginata*	➢*Enterobius*	-----------	-----------
Haematuria	➢*Schistosoma haematobium*	-----------	-----------	➢Plasmodium	-----------
Rectal prolapse	-----------	-----------	➢*Trichuris*	➢	-----------
Appendicitis		➢*T. saginata* ➢*E. vermicularis*	➢*Ascaris* ➢*Trichuris*	➢E. histolytica ➢B. coli	-----------
Ulcer	-----------	-----------	➢*Trichuris* ➢*Ancylostoma* ➢*D. medinensis*	➢E. histolytica ➢B. coli ➢Cutaneous Leishmania ➢Acanthamoeba ➢Microsporidia	➢Siphonaptera (Fleas) ➢Acarina (Ticks) ➢Myiasis

ما كان من خطأ أو نسيان فمنا ومن الشيطان
وما كان من توفيق فهذا من فضل ربنا ليبلونا أنشكر أم نكفر
فالحمد لله حمدا كثيرا طيبا مباركا فيه كما ينبغي لجلال وجهه وعظيم سلطانه
ونسأله سبحانه أن يجعل هذا العمل خالصًا له ليس فيه للشيطان نصيب

لا تنسونا من صالح دعائكم

Best wishes: Your friends & Drs.